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INFARCTION OF THE LATERAL WALL OF THE LEFT VENTRICLE: ELECTROCARDIOGRAPHIC CHARACTERISTICS*†

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PATHOLOGIC studies[2] have shown that, in general, there are three main sites of cardiac infarction: (1) the "anterior apical," (2) the "posterior basal," and (3) the "midventricular," located in the left posterolateral wall of the heart. The electrocardiographic signs of the first two have been established.[3] The present paper has to do with the recognition of the third, the less common, type of cardiac infarction.

Our observations indicate that: (1) Lateral infarction, like the other two, seems to have its own electrocardiographic pattern. (2) This pattern can be mimicked rather closely, in certain cases, by digitalis action. (3) The electrocardiographic features of the acute lesion may subside very rapidly; the tracing during the process of healing may be indistinguishable from that of certain hypertensive patients without cardiac infarction. (4) After healing of the infarction, all the changes produced by it may disappear from the tracing. (5) This group of cases has a very high incidence of auricular fibrillation. (6) Without knowledge of these facts it is possible to make the dangerous mistake of overlooking the presence of this type of acute cardiac infarction.

The electrocardiographic pattern of acute "lateral" or "midventricular" infarction is illustrated by Case 1, Fig. 1*B*. The main features are (1) a depression of the RS-T interval in Lead IV, (2) a depression of the RS-T interval in Leads I and II (commonly, though not universally present), and (3) an absence of the signs of posterior infarction in

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†All tracings in this paper are shown as if they were taken by the technique recommended by the Committee of the American Heart Association for the Standardization of Precordial Leads,[1] and are described in the terminology suggested by it.

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Lead III. In the typical case the RS-T interval depression in precordial leads is more marked when the anterior electrode is placed at or to the left of the apex, than when it is put nearer the sternum (Fig. 2, Cases 2, 3, and 19). Lead III shows left axis deviation in 12 of the 20 cases. However, in five cases (1, 3, 7, 8, and 10) in which former tracings are available for comparison the QRS complex in Lead III is the same before and after the attack. Consequently, left axis deviation cannot be considered an integral part of the electrocardiographic pattern of lateral infarction. One patient with a significant Q_3 is included in this series (Case 10)* because this wave was present prior to the attack in which lateral infarction is thought to have occurred. The QRS complex, not only in Lead III, but in all leads, seems to be singularly unaffected by

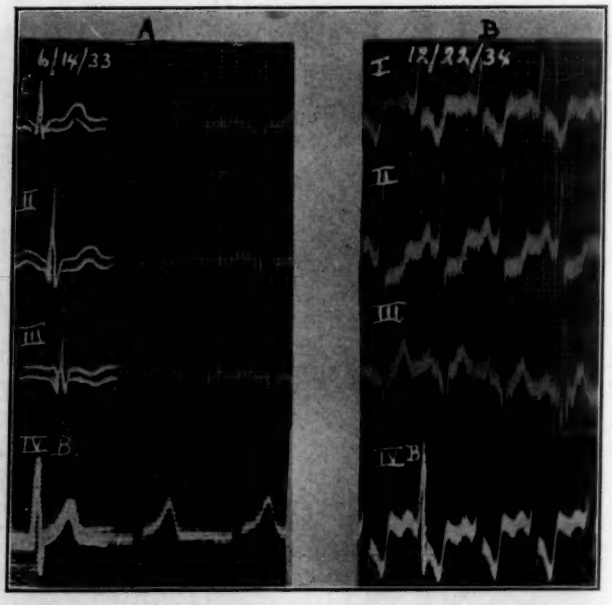

Fig. 1.—Electrocardiograms in Case 1.

In this case and in a number of others, the precordial leads were originally taken with our old technique.[3] However, in order to avoid confusion, they have been rephotographed and printed with the film reversed. Thus, in all figures in this paper, the precordial leads appear as though they had been taken according to the recent recommendations of the Committee for Standardization of Precordial Leads of the American Heart Association.[1]

A, normal tracing taken June 14, 1933, eighteen months before the attack.

B, tracing taken Dec. 22, 1934, nine hours after the attack began, showing evidences of acute lateral infarction. The RS-T interval is depressed in Leads I, II, and IV. Lead III is within normal limits, but shows a marked change in the T-wave since June 14, 1933. The QRS complex has not been changed by the infarction. The patient died ten hours after this electrocardiogram was taken. Necropsy showed a recent infarct in the left lateral wall of the heart.

this type of infarction. The only definite change we have seen was in Case 7 (Fig. 4). In the tracing of this patient the S-wave in Lead IV-B was definitely smaller after the attack than before it.

*Fig. 1 of an earlier paper[4] shows the electrocardiogram in a similar case. A significant Q_3 was present before as well as after an attack which may have been caused by lateral infarction.

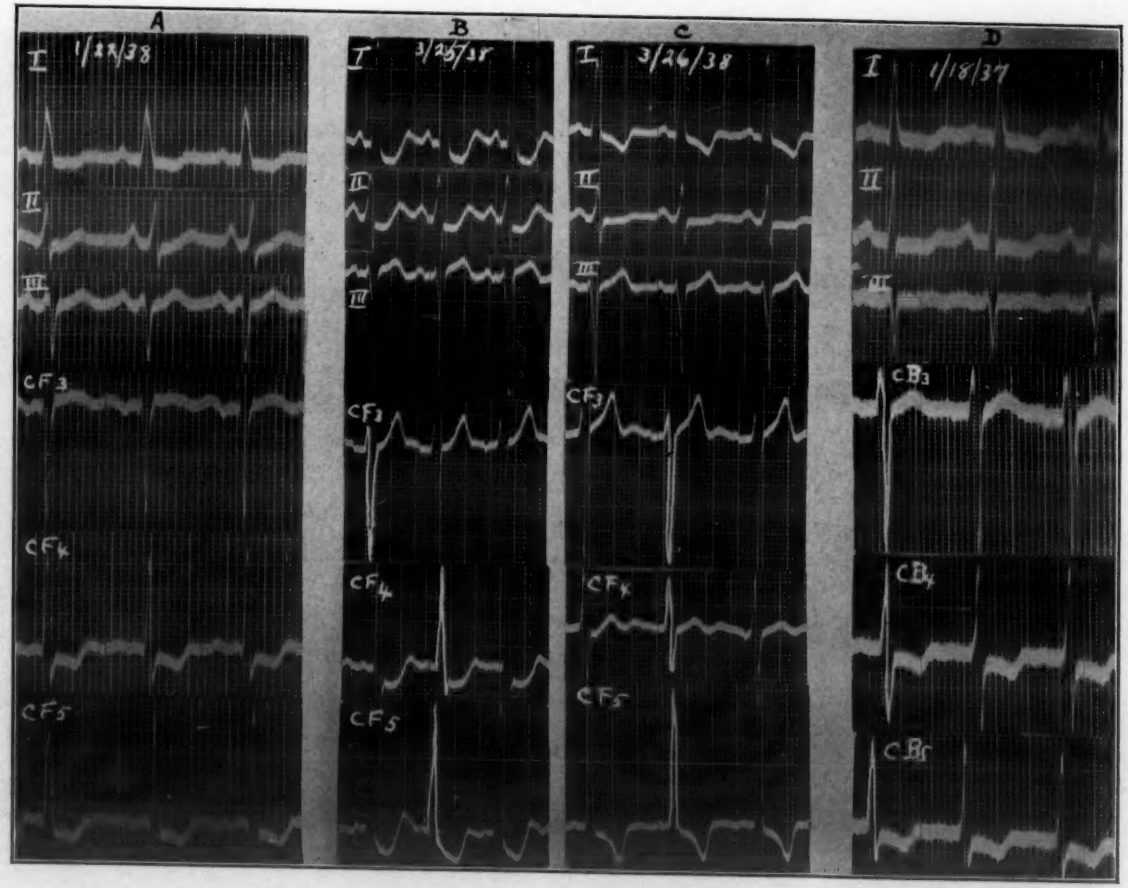

Fig. 2.—*A*, electrocardiogram in Case 2, taken Jan. 22, 1938, seven hours after the onset. The RS-T interval is slightly depressed in Leads I and II. Lead III is normal except for left axis deviation. In precordial leads the depression of the RS-T interval is seen in leads CF_4 and CF_5, but not in CF_3. Necropsy on Jan. 27, 1938, showed recent myocardial damage in the left lateral wall of the heart.

B and *C*, electrocardiograms in Case 19, taken March 25 and 26, 1938. *B*, the tracing taken four hours after the onset of pain, shows the typical pattern of acute lateral infarction. The RS-T interval in Leads I and II is depressed. Lead III is normal except for left axis deviation. In precordial leads the depression of the RS-T interval is marked in CF_4 and CF_5, but CF_3 fails to show it. *C*, a tracing taken twenty-four hours after the attack, shows a complete disappearance of the signs by which the healing infarction could be recognized. The electrocardiogram might well be that of a patient with hypertension without cardiac infarction.

D, electrocardiogram in Case 3, taken Jan. 18, 1937, four days after the last attack of pain. The RS-T interval depressions in Leads I and II are subsiding. Left axis deviation is present. In precordial leads the RS-T interval depression is present in CB_4 and CB_5 but absent from CB_3.

An electrocardiogram almost exactly like that in Case 1 was obtained from a patient during a brief attack of effort angina (Case 21, Fig. 5). Similar evanescent electrocardiographic changes are reported by Jervel[5a] and by Levy, Barach and Bruenn.[5b] It is clear, therefore, that the electrocardiographic pattern of lateral infarction, since it lacks changes in the QRS complex, can be produced by temporary ischemia as readily as by the more permanent circulatory interruption of coronary thrombosis. Furthermore, after the anginal attack the tracing may return completely to normal (Fig. 5*C*).

The electrocardiographic pattern of lateral infarction might be confused with that of posterior infarction on the one hand, or with that of pulmonary embolism[6] on the other, because the precordial leads of all

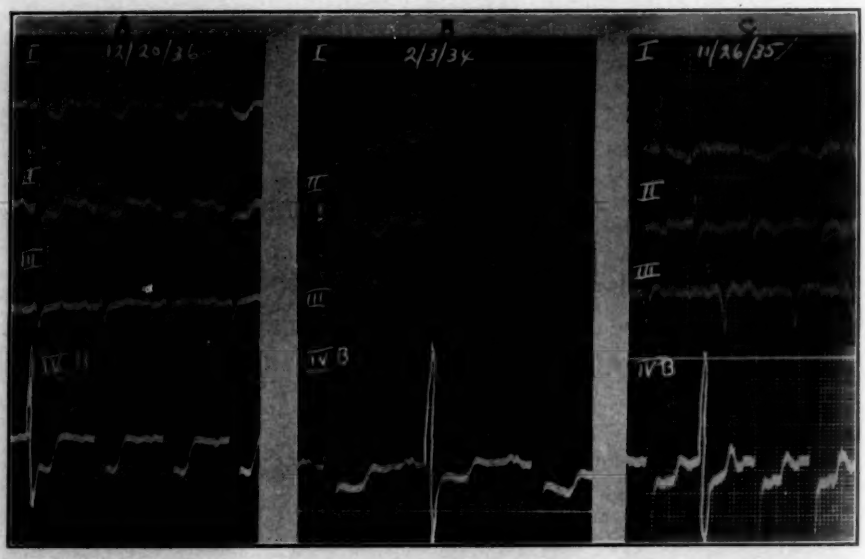

Fig. 3.—*A*, electrocardiogram in Case 4, taken Dec. 20, 1936, twelve hours after the onset. The typical signs of acute lateral infarction are apparent. The RS-T interval in Leads I, II and IV-B is markedly depressed. Lead III is normal except for slight left axis deviation.

B, electrocardiogram in Case 11, taken Feb. 3, 1934, two days after a severe attack of cardiac pain. The signs of recent lateral infarction are present. The RS-T interval in Leads I, II and IV-B is markedly depressed. Slight left axis deviation is present.

C, electrocardiogram in Case 5, taken Nov. 26, 1935, thirty-six hours after the attack. It shows auricular fibrillation, inversion of T_1 and T_2, left axis deviation, and a marked depression of the RS-T interval in Lead IV-B.

three may be similar. However, posterior infarction can usually be differentiated because it has a significant Q_3 and an elevation of the RS-T interval in Leads II and III. Moreover, pulmonary embolism differs in that T_1 is usually upright and T_3 usually inverted.

The evidence that the lesion is lateral or posterolateral in these cases may be summed up as follows:

1. One typical, uncomplicated, autopsy case is available (Case 1). In a second (Case 2) there was no gross infarction at necropsy; but the history of an attack of pain, combined with the finding of recent

thrombosis of a branch of the left circumflex artery and definite histologic changes in the lateral wall of the heart, makes this case a strong link in the chain of evidence. In the third necropsy case (Case 10) there was an old circumflex artery occlusion, but clinicopathologic correlation here is uncertain because of the fourteen-month interval between the attack and the necropsy.

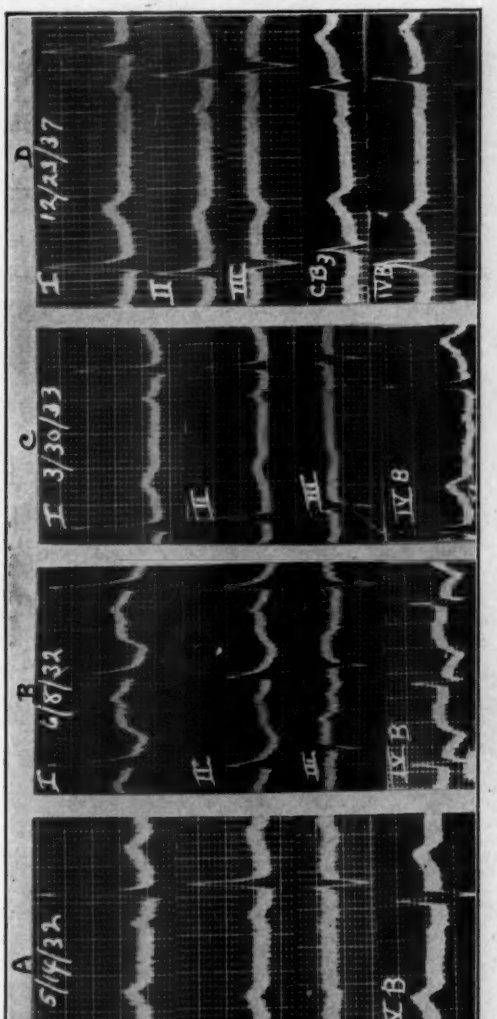

Fig. 4.—Electrocardiograms in Case 7.

A, tracing taken May 14, 1932, three weeks before the attack. It is normal except for left axis deviation.

B, tracing taken June 8, 1932, two hours after the onset of the attack. The RS-T interval is depressed in Leads I, II, and IV. Lead III shows slight left axis deviation, and a slight RS-T interval elevation.

C, tracing taken March 30, 1933, fifteen months after the attack. It is very much like the electrocardiogram of May 14, 1932, prior to the cardiac infarction, except that the S-wave in Lead IV-B has disappeared. This did not take place immediately after the attack, but at some time between June 20, 1932 and March 30, 1933.

D, electrocardiogram taken Dec. 23, 1937, showing that the S-wave in Lead IV-B is still absent. In a lead taken with the precordial electrode nearer the sternum (CB_3), the S-wave is present but much smaller than it was in Lead IV-B before the attack. (Lead CB_3 is ordinarily expected to have a larger S-wave than Lead IV-B.)

2. The electrocardiographic study of infarction in other parts of the heart indicates that myocardial ischemia in a certain location tends to produce a characteristic electrocardiographic pattern. Thus, it is reasonable to suppose that the seventeen cases without necropsy were instances of lateral infarction.

3. This group of cases corresponds in relative size with the group of cases of "midventricular" infarction described pathologically by Barnes and Ball,[2] in that it is smaller than the groups of either anterior or posterior infarction.

4. Even before Case 1 appeared, the electrocardiographic characteristics shown by these patients led us to believe that the lesion was located posterolaterally[4, 7] away from the septum. This belief was based on the fact that (a) Lead IV showed a depression of the RS-T interval (i.e., a posterior rather than an anterior localizing sign), (b) the Q_3 (supposed to be due to a lesion in or near the posterior basal portion of the interventricular septum) did not appear, and (c) the RS-T interval elevation in Lead III, so characteristic of typical posterior infarction, was absent.

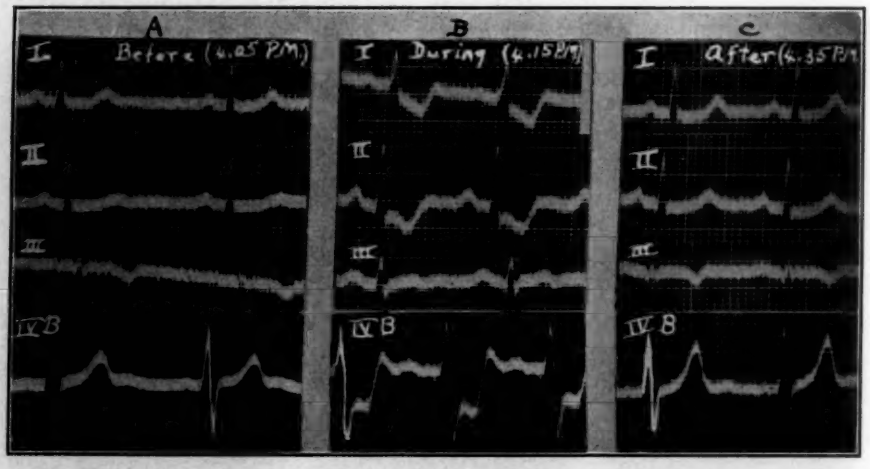

Fig. 5.—Tracings in Case 21, taken May 19, 1934, which show that the typical electrocardiographic pattern of acute lateral infarction can appear temporarily during an attack of effort angina.

A, taken at 4:05 P.M., before exercise, showing no definite abnormalities. The subject then induced lower sternal discomfort of moderate degree by stepping up on a chair twenty-five times, and then swinging his arms for a brief period.

B, taken at 4:15 P.M., during the height of his discomfort. The patient classified this attack as of only moderate severity, compared with others he had experienced. The typical pattern of acute lateral infarction has appeared. The RS-T interval in Leads I, II, and IV has become markedly depressed. The deviation in RS-T IV is the largest we have ever seen during an attack of effort angina.

C, taken at 4:35 P.M., after the discomfort had subsided. The tracing has returned to normal, and looks much like it did before the attack. The evidence suggests that this patient had ischemia of the left lateral wall of the heart during this attack of effort angina.

Final acceptance of the fact that this electrocardiographic pattern is caused by lateral infarction should probably await the report of other necropsy cases, uncomplicated by digitalis medication or by multiple myocardial lesions. However, all available evidence would seem to favor this point of view.

Atypical Cases.—Midventricular or lateral infarction usually results from occlusion of the circumflex branch of the left coronary artery.[2]

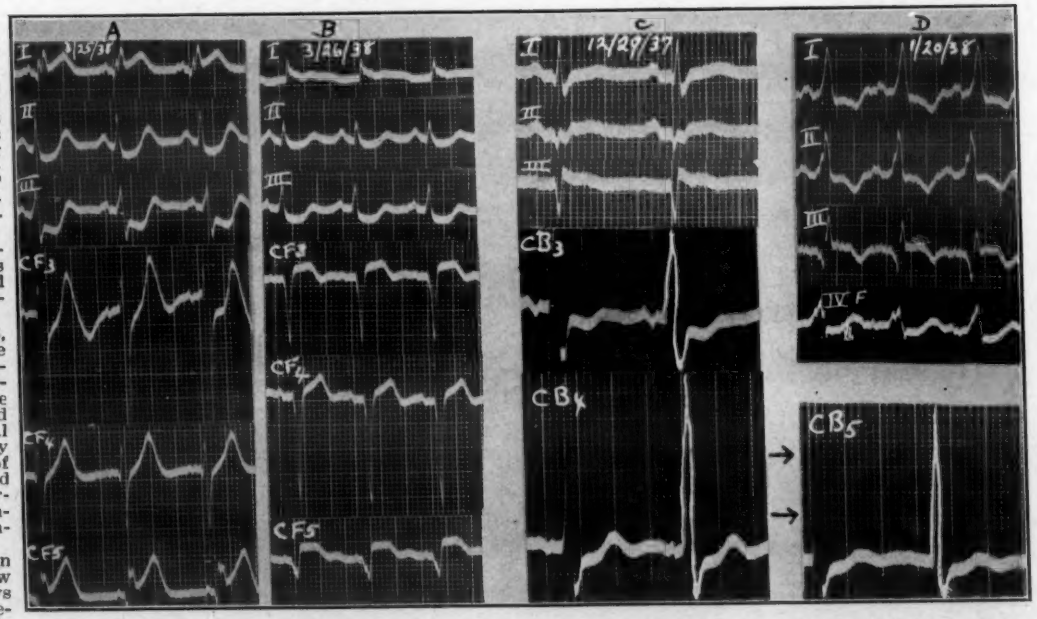

Fig. 6.—Atypical cases.

A and *B*. Electrocardiograms in Case 22.

A, tracing taken March 25, 1938, on the day of onset. The limb leads suggest anterior infarction. The precordial leads seem superficially contradictory. CF_3 shows an RS-T interval depression, suggesting lateral or posterior infarction; CF_4 is normal; CF_5 shows a Q-wave and an RS-T interval elevation. Anterolateral infarction was suggested ante mortem as an explanation for this electrocardiogram.

This tracing illustrates the marked differences which may occur in certain cases between Leads CF_3, CF_4 and CF_5. All three may be worth taking in certain patients with coronary disease.

B, tracing taken in Case 22, March 26, 1938, showing the typical pattern of acute anterior infarction in limb leads. The precordial leads have changed markedly, suggesting an enlargement of the lesion in the anterior wall of the left ventricle. Lead CF_5 shows the most marked RS-T interval elevation of any precordial lead. Necropsy March 28, 1937, showed old occlusion of the left anterior descending artery, and recent occlusion of the left circumflex artery with extensive infarction of the anterior and lateral walls of the left ventricle.

C, electrocardiogram in Case 24 taken Dec. 29, 1937. Leads II and III show RS-T interval elevation. Lead III shows a Q-wave. Precordial leads show a depression of the RS-T interval, which in this case is as marked in CF_3 as in CF_4 and CF_5. Necropsy Jan. 3, 1938, revealed old occlusion of left anterior descending and right coronary arteries, with recent occlusion of the left circumflex. There was an extensive infarct involving the posterior and lateral walls of the left ventricle.

D, electrocardiogram in Case 23, taken Jan. 20, 1938, showing evidences of recent lateral infarction (i.e., an RS-T interval depression in Leads I, II, and IV) combined with the signs of fresh posterior infarction (i.e., a significant Q-wave and an RS-T interval elevation in Lead III). Necropsy Jan. 24, 1938, showed a small patent right coronary artery, an old occlusion of the left anterior descending artery, and a recent occlusion of the large left circumflex. There was extensive infarction of the posterior and lateral walls of the left ventricle, and of the posterior part of the interventricular septum.

However, in a heart with an unusually large left circumflex or with neighboring arteries congenitally small or obstructed by disease, occlusion of this vessel may give rise to a more extensive lesion which may invade the anterior or the posterior wall of the left ventricle. Thus complex electrocardiographic patterns may be produced. Four such cases have been seen by us, each with necropsy (Cases 22 to 25, inclusive). Case 22 is an example of circumflex occlusion which produced infarction extending to the anterior wall because of former occlusion of the left anterior descending artery. The anterolateral location of the lesion was suggested by the tracing obtained on March 25, 1938, (Fig. 6*A*).* The other three are examples of circumflex occlusion with extension of infarction into the posterior wall of the left ventricle. In all three the circumflex artery was unusually† large and the other coronary arteries were either unusually small (Case 25), or obstructed as a result of previous disease (Cases 23 and 24). The electrocardiogram in Case 23 (Fig. 6*D*) is fairly typical of lateral infarction except that Lead III has the characteristics of posterior infarction. In Case 24 (Fig. 6*C*) the chest leads suggest lateral or posterior infarction, but the RS-T interval is elevated in Leads II and III, and Q_3 is present. In Case 25 there is a tendency to right axis deviation (the patient had mitral stenosis); the T-waves in the limb leads are normal; a definite RS-T interval depression is present in Lead IV-B.

In the study of our cases of lateral infarction, three peculiarities have been encountered which have made the diagnosis difficult:

I. There is a high incidence of auricular fibrillation. If we include the four patients with infarction extending beyond the "midventricular" region, the group totals 24. Ten of these showed auricular fibrillation at some time during their course. One (Case 9) had a 48-hour paroxysm following prostatectomy, 9 weeks after his coronary occlusion. Two patients (Cases 6 and 8) probably had fibrillation before, as well as after, the attacks we studied. In a fourth (Case 18) the time of onset of the fibrillation is not definitely known. However, since the arrhythmia ceased a few days after admission, it was probably a paroxysm accompanying the coronary attack. In a fifth (Case 25) mitral stenosis was present and may have predisposed the patient to the occurrence of auricular fibrillation. The sixth patient (Case 10) had a good many paroxysms of auricular fibrillation, often accompanied by cardiac pain. On April 24, 1933, when we suspect that a lateral infarction occurred,

*For some time it has been obvious that in certain cases of coronary occlusion a single chest lead is inadequate to elicit all available diagnostic information. Case 22, Fig. 6*A*, is an example of the differences which sometimes exist between tracings taken with the anterior electrode at three different points on the precordium. On account of the findings in cases of this sort, we are, at present, using three precordial leads, CF_2, CF_4 and CF_5[1], in all patients suspected of having coronary disease.

†"Unusually" may be too strong a term. Gross[8] has shown that in about one of every ten individuals the circumflex branch of the left coronary artery is a large vessel which continues on to form the posterior descending artery, and supplies the entire posterior surface of the left ventricle. In hearts in which this anatomical situation exists (Cases 23 and 25), the "atypical" vascular pattern may regularly produce an "atypical" electrocardiographic pattern when the left circumflex artery is occluded.

he had an especially long paroxysm. The four other patients (Cases 2, 22, 5, and 17) developed fibrillation at the time of their cardiac infarction. In the first two the rhythm returned to normal, and in the last

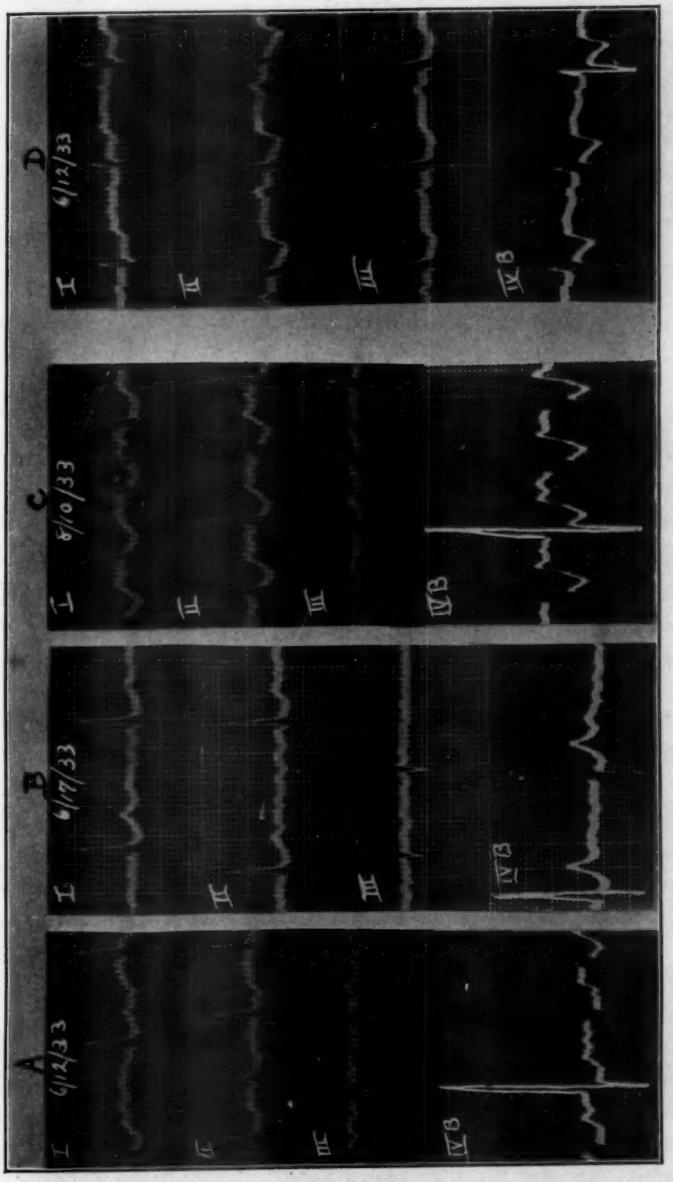

Fig. 7.—Digitalis effects.

A, *B*, and *C*, are electrocardiograms in Case 17. They all show auricular fibrillation, which began at the time of the attack, June 8, 1933. *A* is the tracing taken June 12, 1933. It has a suggestion of a depression of the RS-T interval in Leads I and II, and a definite depression of this interval in Lead IV-B. *B* is the tracing taken June 17, 1933, which shows a return of the RS-T interval deviations towards the isoelectric line. *C* is the tracing taken Aug. 10, 1933, when clinical evidences suggested that the infarct had healed. The patient was fully digitalized at this time. The typical electrocardiographic pattern of acute lateral infarction is present.

D is the tracing of a man of 50 years without demonstrable heart disease. On May 24, 1933, his electrocardiogram was normal. Beginning May 25, 1933, he received 6 c.c. of the tincture of digitalis daily. On June 12, 1933, the electrocardiogram shown in *D* was taken. It has a depression of the RS-T interval in all four leads, the usual result of digitalis action when the electrical axis is normal.

When digitalis was stopped, the tracing returned to its original configuration.

two the arrhythmia persisted. Thus, the evidence suggests an etiologic relationship between the attack of pain and the onset of auricular fibrillation in at least four cases, and probably in seven. In the remaining three cases, it is not impossible that the arrhythmia was related to the coronary lesion.

As a background for comparison, the records of 124 unselected cases of coronary occlusion were reviewed. Only six patients had auricular fibrillation. Dividing these cases into groups shows the following: In 64 cases of anterior infarction, there were 5 with auricular fibrillation, all with relatively brief paroxysms. One of these patients also had an attack of auricular flutter. Among 10 patients suspected of having anterior and posterior infarction,[9] one had a short paroxysm of fibrillation. In the remaining 50 cases, 37 with posterior infarction, 11 with huge T-waves in chest leads,[10] and two with infarction of doubtful location, there was no instance of auricular fibrillation. Thus, no matter how the figures of the cases of lateral infarction are interpreted, whether there are 10 of 24, 7 of 24, or only 4 of 24 with a definite relationship between the coronary attack and the arrhythmia, the incidence is higher than that of its nearest rival (1 in 10), and much higher than that of the entire coronary group (1 in 20).

The cause for this high incidence of auricular fibrillation in lateral infarction is not known. It may be that the auricular muscle is deprived of a necessary part of its blood supply by circumflex occlusion. Digitalis could not have been an important factor since 7 of the 10 patients received none of this drug prior to the onset of arrhythmia.

II. Digitalis administration may produce an electrocardiographic pattern similar to that of lateral infarction.[11] Because of the high incidence of auricular fibrillation, this factor comes into play with a frequency unparalleled in other types of coronary occlusion. In patients with a normal electrical axis, with the main deflection of QRS in Lead III directed upward, digitalis depresses the RS-T interval in all four leads (Fig. 7*D*), and gives a little diagnostic difficulty. In patients with left axis deviation, digitalis depresses the RS-T interval in Leads I, II, and IV, and elevates it in Lead III. Thus, with mild degrees of left axis deviation the effect of this drug might closely mimic the lateral occlusion pattern. In only one type of case, however, have we seen digitalis reproduce exactly the picture of acute lateral infarction, i.e., in a patient with a healed lateral infarct. This phenomenon has been observed in other types of coronary occlusion.[12] The patient in Case 17 (Fig. 7*C*) is probably an example. His attack occurred June 8, 1933. RS-T interval deviations, which were present June 12 (Fig. 7*A*), began to subside June 17 (Fig. 7*B*). The lesion had probably healed fairly well by Aug. 10, 1933, as far as could be determined on clinical grounds. However, at this time, when the patient was fully digitalized, the tracing shows the most marked RS-T interval deviations with the typical picture of acute lateral infarction (Fig. 7*C*). Other examples of this phenomenon are the electrocardiogram of the patient in Case 16, taken on March 16, 1936, and possibly the tracings in Case 8 (Nov. 23, 1933), Case 13 (Dec. 19, 1932) and Case 15.

This brings up the question whether, in this group of cases, some or all of the electrocardiographic features which we have attributed to lateral infarction might have been due to digitalis. The facts are these: Prior to the taking of the first, or at least the significant electrocardiogram, 5 patients were probably digitalized though free from toxic effects (Cases 6, 8, 9, 13 and 15); 2 had received 12 cat units or less (Cases 7 and 18); 2 had received less than 6 cat units (Cases 2 and 17); 11 had received no digitalis at all. Thus the chances that any but the first five had received enough of the drug to cause the electrocardiographic changes would seem to be meager. Moreover, even in them it is hardly proper to attribute the electrocardiographic features to digitalis because (1) comparatively few patients have marked RS-T interval deviations from digitalis without toxic symptoms, such as nausea and vomiting, or marked asthenia, (2) few digitalized patients show the exact pattern of lateral infarction, and (3) all five of our patients had attacks of pain which suggested coronary occlusion.

Nevertheless, when confronted with an electrocardiogram suggesting recent lateral infarction, especially if left axis deviation is present, one should remember that digitalis can, very nearly, reproduce the pattern. Moreover, in old people with myocardial disease, much less of the drug is required to cause RS-T interval deviations than is necessary in young healthy subjects, and the effects in this older group persist much longer than in young individuals.

III. After RS-T interval deviations disappear the lesion becomes unrecognizable electrocardiographically. In seven patients repeated tracings were taken during the healing of the infarct. After the attack the RS-T interval reached the isoelectric line within the following time intervals: Case 19, 24 hours; Case 7, 2 days; Case 10, 3 days; Case 20, 7 days; Case 3, 13 days; Case 14, 21 days; and Case 11, 28 days; although the typical pattern, by means of which the diagnosis could be readily established, often became indistinct sooner than this. At the end of these periods of time, the electrocardiogram usually showed an inversion of T_1 and T_4, a diphasic T_2 and an upright T_3. When left axis deviation was present (6 of 7 cases), the tracing could not be distinguished from the pattern often seen in patients with hypertension without cardiac infarction.

Nine patients, whose last tracings were free from digitalis effects, were followed electrocardiographically for a month or more, i.e., into the "healed stage." Three different end results were observed. (a) Five retained the hypertensive-like pattern described above (Cases 3, 8, 11, 13, and 14), (b) one showed a deep Q_3 and an inverted T_3 (Case 10), and (c) the other three showed electrocardiograms which were normal except for left axis deviation (Cases 7, 9, and 20). In four of the nine (Cases 3, 7, 8, and 10), tracings made before the attack are available for comparison. In every one of the four, the electrocardiogram after

healing resembles very closely the tracing taken before the attack except for a reduction of S_4 in Case 7.

Thus the evidences of acute infarction may disappear rapidly, leaving a partly healed lesion with no diagnostic electrocardiographic signs. Moreover, all evidences that an infarct ever occurred may disappear after healing; the tracing usually returns to its former contour; if it was normal originally, the final electrocardiogram may be normal.

This is not unexpected when one considers the electrocardiographic behavior of infarcts in general. When infarction heals: (a) The QRS complex changes which it produced tend to persist, but lateral infarction has none of these. (b) The T-wave in each lead tends to assume a direction opposite to that of the former RS-T interval deviation, but in acute lateral infarction the RS-T interval deviations are opposite in direction in each lead to normal T-waves. Consequently, they tend to give place to normal T-waves with the passage of time.

DISCUSSION

For a good many years lateral infarction was a confusing factor in the electrocardiographic study of coronary occlusion. Occasionally we saw patients with this type of tracing who gave a history of cardiac pain. However, we saw patients with similar electrocardiograms in whom auricular fibrillation was present, and to whom digitalis had been administered. The first definite clue came in December, 1934, when the patient described as Case 1 appeared and demonstrated that this electrocardiographic pattern could be caused by infarction of the lateral wall of the left ventricle, in the absence of auricular fibrillation or digitalis medication. The second clue was obtained when all cases in which cardiac pain was accompanied by this type of tracing were reviewed. Then the unexpected fact appeared that in this group of cases of coronary occlusion the incidence of auricular fibrillation was high. The third step was to review all cases with electrocardiograms of this sort in which the pattern of the tracing had been attributed to digitalis administration. Then it was discovered that all patients with electrocardiograms like that shown in Fig. 1*B* had clinical evidences suggestive of recent coronary occlusion.

The establishment of the fact that a lateral infarct may cause this type of electrocardiogram has removed an important source of diagnostic confusion. On the basis of electrocardiographic study during the acute stage, most large infarcts may now be classified as anterior, posterior, lateral, or combinations of these types. However, the establishment of the electrocardiographic pattern of lateral infarction has accomplished more than this. By making possible the study of a group of these cases, it has elucidated certain pitfalls in diagnosis, knowledge of which is essential if the costly mistake of overlooking a coronary thrombosis is to be avoided.

In three types of patients with lateral infarction there is very real danger of failing to recognize that coronary occlusion has occurred. (1) The case which is most confusing is that in which the lateral infarction is accompanied by auricular fibrillation, and digitalis is given before the electrocardiogram is made. The precordial discomfort is likely to be attributed to the arrhythmia, and the RS-T interval deviations to digitalis medication. (2) Another case which might give rise to dangerous misinterpretation is that of the individual with lateral infarction whose first electrocardiogram is not made until several days after the attack. If RS-T interval deviations have subsided and the tracing looks like that of a patient with hypertension, the original clinical impression of coronary thrombosis may be shelved while a dangerous search for a "better" diagnosis is conducted. (3) A third condition which might be misjudged is that of the patient who, three months after an attack of lateral infarction, is found to have an electrocardiogram which is normal except for left axis deviation. It would require a very typical clinical history to offset this negative finding in the minds of a good many physicians.

In the past six years we have come to lean quite heavily upon the electrocardiograph in the diagnosis of coronary disease. Because anterior and posterior infarctions often have characteristic signs during the acute, healing, and healed stages, we have been led to expect to find diagnostic, or at least suggestive, electrocardiographic findings after all major attacks of coronary thrombosis. This group of cases of lateral infarction shows that if the electrocardiographic method is leaned upon too heavily in diagnosis, it may prove a broken reed. The physician may obtain from it an unjustified, dangerous sense of security.

SUMMARY

Evidence is presented in this paper which suggests that acute infarction in the left lateral wall of the heart produces in the electrocardiogram a depression of the RS-T interval in Lead IV and usually a depression of this interval in Leads I and II. Lead III shows no characteristic abnormalities.

The RS-T interval deviation in Lead IV is often more marked when the precordial electrode is placed at or to the left of the apex than when it is put nearer the sternum.

The QRS complex is usually unaffected by lateral infarction. Consequently, the electrocardiographic pattern of this lesion can be reproduced by angina of effort.

Left circumflex artery thrombosis is usually responsible for infarction in the left lateral wall of the heart. However, when this vessel carries an unusually large part of the myocardial blood supply, its obstruction may give rise to more extensive infarction, with complex electrocardiographic patterns.

In some patients with lateral infarction there is very real danger of failing to recognize the fact that a coronary occlusion has occurred because (a) these patients frequently have auricular fibrillation, (b) digitalis action may produce a somewhat similar tracing, and (c) electrocardiographic signs of the lesion may disappear rapidly and completely.

The electrocardiographic method is a much less sensitive diagnostic procedure in lateral infarction than it is in anterior or in posterior infarction. Even when several precordial leads are used, a lesion in the left lateral wall of the heart, though incompletely healed, can escape detection.

On the basis of electrocardiographic study during the acute stage, it is now possible to classify most large infarcts as anterior, posterior, lateral, or combinations of these three types.

CASE REPORTS

CASE 1.—B. Z., a white man aged 54 years, was admitted to the Mount Sinai Hospital Dec. 22, 1934. He had suffered from effort angina for two years. A few days before admission, the attacks had increased in frequency and severity. At 3 A.M. on the day of admission a substernal and precordial pain began which radiated to both shoulders and both arms. It was worse than any he had ever had before. The pain was not relieved by the repeated administration of nitroglycerin, and only slightly ameliorated by two doses of pantopon.

On admission, at 12:45 P.M., the blood pressure was 88/66 (it had been 150 systolic prior to the present attack); the temperature was 97.8° F.; the pulse rate was 110 per minute; the respiratory rate was 32 per minute. The patient was dyspneic and cyanotic; the heart sounds were distant; coarse râles were heard in both lungs. The leucocyte count was 22,500 per cu. mm., and 88 per cent of the leucocytes were neutrophiles. The patient became steadily worse, developed pulmonary edema, and died at 11 P.M.

A necropsy was performed Dec. 23, 1934, by Dr. Meranze. The heart weighed 390 gm. It was normal in size and position. The left anterior descending coronary artery was atheromatous but had an "adequate lumen." The right coronary artery, though rigid, had a "good lumen." The left circumflex artery immediately after its origin showed marked reduction of caliber from former disease. At the point of narrowing, the remainder of the lumen was occluded by a red thrombus.

The heart muscle was flabby. There were fibrotic areas in the anterior surface of the left ventricle near the apex. A fresh infarct measuring 6 by 3 cm. was found in the "mid lateroposterior region of the left ventricle."

Two electrocardiograms are available (Fig. 1): *A*, a tracing taken June 14, 1933, eighteen months before death, which is essentially normal, and *B*, an electrocardiogram taken Dec. 22, 1934, shortly after admission, which shows marked R-ST interval deviations. The characteristics of this tracing differ from those seen in either anterior or posterior infarction. Lead IV-B shows a marked RS-T interval depression and a normal QRS complex. Leads I and II show a marked depression of the RS-T interval. Lead III shows a relatively normal ventricular complex without a significant Q-wave or a displacement of the RS-T segment. This patient received no digitalis.

CASE 2.—S. M., a white man 72 years old, with a history suggesting a coronary attack in 1936, came to the University of Pennsylvania Hospital Jan. 11, 1938, for lower abdominal symptoms. Dr. Ravdin operated to relieve a colonic obstruction

on Jan. 20, 1938. At 4 A.M. on Jan. 22, 1938, the patient had a severe attack of substernal pain. The heart became rapid (rate 180) and totally irregular. Morphine and 4 grains of digitalis were prescribed, and the patient was placed in an oxygen tent. The arrhythmia subsided in two hours. The pain gradually wore off, leaving a soreness. The patient felt "knocked out" afterwards, but the blood pressure did not fall. The significance of fever and leucocytosis was uncertain because the attack occurred during the postoperative reaction. There were several recurrences of pain on January 25 and 26, and the blood pressure fell from 200/100 to 90/55. Death, apparently cardiac, took place Jan. 27, 1938.

A necropsy was performed by Dr. Lippincott on the day of death. The left anterior descending coronary artery showed an organized obstruction, partly canalized, 6 cm. from its orifice. The right coronary artery was patulous but small and did not reach the posterior interventricular groove. The main channel of the left circumflex was patulous. One of its branches, low on the posterolateral surface of the left ventricle, was obstructed by a brown thrombus of recent origin.

The myocardium showed an area of healed infarction in the lower lateral surface of the left ventricle, with several small areas of fibrosis anterior to it. Since no definite recent infarction was visible grossly, a number of sections were cut from various portions of the left ventricular wall. On microscopic examination healed infarction was found in the areas where it had been visible to the naked eye. Recent degeneration of muscle fibers was found in all three sections taken from the lateral wall of the left ventricle. Some suggestion of recent degeneration was found in the anterior wall of the left ventricle, but not much. None was seen in the interventricular septum or in the posterior wall nearby.

No possible cause of death except the cardiac lesion was found.

Electrocardiograms were taken on January 22 (Fig. 2*A*), January 24 and 25, 1938. All were similar and resembled those in Case 1, except that left axis deviation was also present. The RS-T interval depression in chest leads CF_4 and CF_5 was not seen in lead CF_3.[1]

CASE 3.—J. S., a white man 53 years old, had attacks of severe substernal and upper abdominal pain lasting two hours each on Jan. 12 and 14, 1937. There were minor seizures in the interim. The blood pressure, known to have been 225/125 on Dec. 1, 1936, dropped to 140/70 after the attack. The blood sedimentation rate, which had been 10 mm. in one hour Dec. 2, 1936, became 24 mm. in one hour on Jan. 15, 1937. There was slight fever after the attack, but no leucocytosis. The clinical diagnosis was coronary thrombosis. The patient recovered, returned to work, and on Feb. 4, 1938, was reported to be alive and well.

Many electrocardiograms are available. One taken Dec. 2, 1936, while he was in the University of Pennsylvania Hospital on account of a small cerebral thrombosis, before he had had any cardiac pain, shows an inversion of T_1, a diphasic T_2, an upright T_3, an inverted T_4, left axis deviation, and no RS-T interval deviations. Electrocardiograms after the attack, shown in Fig. 2*D*, resemble those in Case 1. By Feb. 12, 1937, all RS-T interval deviations had disappeared, and the tracing had reverted to its former appearance. No digitalis was given to this patient.

CASE 4.—(This case was observed by Dr. E. Bruce Brooks, of Winston-Salem, N. C., and is reported here by reason of his courtesy.) Mrs. J. E. C., a woman 66 years of age, who had hypertension and angina of effort, suffered a severe attack of cardiac pain during the night of Dec. 19, 1936. Morphine was required. The blood pressure dropped from 200/120 to 90/70. Death occurred Dec. 24, 1936. Permission for necropsy was not obtained. Two electrocardiograms were taken, one on Dec. 20, 1936 (Fig. 3*A*), the other on Dec. 21, 1936. The patient did not receive digitalis.

CASE 5.—(This case was observed by Dr. Alexander Margolies, who has kindly permitted us to report it.) F. L., an ex-professional baseball player 57 years old, had had hypertension (160 to 210 systolic) for at least two years. On Nov. 11, 1935, while driving his car, he experienced pain in both arms and elbows. On the night of Nov. 24, 1935, a similar but much more severe attack occurred, accompanied by pain under the sternum and dyspnea. Morphine was administered. The next day the pain disappeared; the temperature was 99.6° F.; gallop rhythm was heard; an electrocardiogram (Fig. 3*C*) showed auricular fibrillation (which had not been present before the attack) and ventricular complexes like those in Case 1, with left axis deviation. The fever persisted for a few days. The blood pressure gradually fell to 150/100. The arrhythmia continued. The patient was digitalized after the electrocardiogram was taken. The clinical diagnosis was coronary thrombosis complicated by auricular fibrillation. On Dec. 19, 1935, the patient died suddenly. Permission for necropsy was not obtained.

CASE 6.—This case has been reported previously and the electrocardiogram has been published (Case 1, Fig. 24[12]). The patient was a woman 47 years of age who had hypertension and who developed auricular fibrillation in October, 1932. In January, 1933, she experienced several severe attacks of epigastric pain and an embolus lodged in her right ulnar artery. She died at home in February, 1933. The clinical picture was very suggestive of coronary occlusion, but on account of digitalis administration, and an "atypical" electrocardiogram, the diagnosis was uncertain. We now believe that she probably had a lateral infarction. Her digitalis dosage was relatively small. From Dec. 30, 1932, to Jan. 4, 1933, she received 1 cat unit t.i.d. From Jan. 4 to 18, 1933, she received 1 cat unit daily. All four electrocardiograms taken, Jan. 9, 12, 14, and 16, 1933, showed auricular fibrillation and ventricular complexes like those in Fig. 1*B*, with left axis deviation in addition.

CASE 7.—R. C., a white woman 48 years of age, came to the Hospital of the University of Pennsylvania May 13, 1932, on the service of Dr. George P. Muller. After careful study, including an electrocardiogram (Fig. 4*A*) and orthodiagram, we were uncertain whether she had gall bladder disease, coronary disease, or both. She gave a history of an attack suggesting a small coronary occlusion in 1930, after which she had substernal pain on effort. Cholecystectomy was done May 17, 1932. Gallstones were found. Convalescence was uneventful. On June 2, 1932, and again June 5, the patient experienced brief periods of syncope and precordial pain. Ten cubic centimeters of tincture of digitalis were administered between June 2 and 8, 1932, after which no more was given. On June 8, 1932, after lunch she had a major attack of pain over the heart which was referred to the left arm and was accompanied by vomiting, cyanosis, dizziness, and a cold sweat. After an hour, morphine was given and brought relief. The next day the leucocyte count was 11,200. Slight fever was noted occasionally from June 3 to 9. After the attack there was no definite change in blood pressure, pulse, or respiration. Gradual improvement took place, and the patient was discharged June 22, 1933. She has been followed in the Cardiac Clinic since that time. When last seen, Dec. 23, 1937, she had definite angina of effort and the blood pressure was 155/110, but there were no objective evidences of cardiac damage.

Many electrocardiograms were taken. The tracing before the attack (Fig. 4*A*) is normal except for left axis deviation. That on the day of the attack (Fig. 4*B*) resembles Fig. 1*B*. Within the first thirty-six hours the signs of acute infarction subsided, leaving T_1 and T_2 diphasic, T_3 flat, and T_4 inverted. Subsequent tracings (Fig. 4*C* and 4*D*) show a permanent reduction in the size of the S-wave in precordial leads. Otherwise the electrocardiogram looks just as it did before the attack. This reduction of the S-wave in Lead IV did not immediately accompany the acute attack. It occurred at some time between June 20, 1932, and March 30, 1933.

CASE 8.—G. S., a woman of 57 years, with a history of angina pectoris, came into the University of Pennsylvania Hospital Nov. 15, 1933, with hemoptysis and auricular fibrillation. There is no record of cardiac pain on this admission. She was digitalized. An electrocardiogram made Nov. 23, 1933, resembles that in Case 1, Fig. 1, except that left axis deviation was present. No precordial leads were taken. On Jan. 25, 1934, when she was readmitted because of glaucoma, she still had auricular fibrillation. From Jan. 27, 1934, to Feb. 4, 1934, 24 grains of powdered digitalis were administered. It was then stopped. On Feb. 5, 1934, the patient had a prolonged severe attack of precordial pain. The blood pressure dropped from 140/100 to 120/60. The leucocyte count rose from 7,600 on Jan. 26, 1934, to 15,000 on Feb. 7, 1934. The temperature was one degree above normal three times during the next few days. A consultant diagnosed coronary thrombosis.

The electrocardiogram made Feb. 6, 1934, resembles that in Fig. 1*B*, but because of digitalis medication it was not reported as showing evidence of coronary thrombosis. The patient improved and was discharged.

On May 1 and June 11, tracings were taken when digitalis effects were absent. T_1 was low, T_2 and T_3 were upright. Left axis deviation and auricular fibrillation were present. Chest leads were not taken.

The patient was last seen March 7, 1938. At that time an electrocardiogram showed auricular fibrillation, left axis deviation, more slurring of QRS, an inverted T_1 and normal T-waves in all other leads. Cardiac pain was not an outstanding symptom at this time because her activity was limited by her poor eyesight.

CASE 9.—J. B., a white male of 66 years, had experienced precordial pain for several months. On Dec. 9, 1933, he came to the University Hospital for a prostatectomy by Dr. Alexander Randall. Digitalis in doses of 1½ grains t.i.d. was started on the day of admission and was continued until December 27. On December 13 the patient had a severe precordial pain which lasted all night. The blood pressure dropped from 160/90 to 135/80, and later to 104/60. Cyanosis, fever, leucocytosis, and right basal râles appeared, and the patient looked "knocked out." He recovered, had his prostate removed Jan. 30, 1934, experienced an attack of auricular fibrillation from Feb. 2 to 4, 1934, and was discharged February 24. When last seen, Nov. 4, 1935, he had had cardiac pain after chopping wood, and showed a few extrasystoles.

Many electrocardiograms were taken. The first, Dec. 20, 1933, during the acute stage, looked like Fig. 1*B* with less RS-T interval deviation in Leads I and II and with left axis deviation. On Dec. 27, 1933, RS-T interval deviations were more pronounced. By Dec. 29, 1933, they had subsided markedly, and on Jan. 2, 1934 they had disappeared. The tracing was normal except for left axis deviation on Jan. 10, 1934, and Jan. 22, 1934. On Feb. 4, 1934, it showed auricular fibrillation with a small T_1 and T_2 and inverted T_3. On April 17, 1934, and Nov. 4, 1935, it was normal except for left axis deviation.

CASE 10.—W. C. was a white male who developed angina pectoris in 1918 at the age of 50 years; it continued until his death, June 30, 1934, at the age of 66 years. At necropsy, July 1, 1934, the left anterior descending coronary artery was small but patent. The lumen of the right coronary artery was obliterated at two different points by organized thrombi: the first was 2 cm. from its orifice, and the second was in the posterior descending artery. The left circumflex artery was large. Its lumen was occluded 2 cm. from its origin by an organized thrombus. No fresh thrombi were found in the coronary arteries. The left ventricular wall at the extreme apex was very thin and fibrous. The ventricular septum and the posterior wall of the left ventricle showed marked scarring, undoubtedly as the result of a former infarct. The whole heart was flabby. The posterior surface looked more reddish and felt softer than the rest. The heart weighed 540 gm.; its chambers were dilated; there were no valve lesions.

From the time when this patient first came under our care, Jan. 8, 1931, until his death, he kept in close contact with us, and many electrocardiograms were taken, probably about fifty in all. From the standpoint of the present paper, the following facts should be mentioned: (1) When he first appeared there was a deep Q_3 in his electrocardiogram. He probably had a healed posterior infarct at that time. (2) He experienced repeated paroxysms of auricular fibrillation. The first occurred in September, 1931, and brought on anginal pain. On Jan. 13, 1934, he had a paroxysm without cardiac pain. On May 10, 1934, another attack of auricular fibrillation occurred without anginal pain except during effort. The pain was relieved by nitroglycerin and rest, though the arrhythmia persisted. (3) He experienced three major attacks of cardiac pain which might have been due to cardiac infarction. The first occurred Nov. 24, 1932. It lasted eleven hours and was accompanied by auricular fibrillation. The patient was hospitalized until Jan. 16, 1933, and treated for coronary occlusion. Electrocardiograms showed a marked RS-T interval depression in chest leads and suggestions in the limb leads of fresh posterior infarction. The second is the one during which we believe the circumflex thrombosis may have occurred. On the morning of April 24, 1933, after having had many severe anginal attacks in the preceding twenty-four hours, he was seen in the cardiac clinic. The rhythm was normal. Nothing definitely new was found, and he was sent home to rest. At 1 P.M. very severe anginal pain began which was not relieved by three doses of nitroglycerin. He was brought to the Hospital of the University of Pennsylvania that afternoon, pale, cyanosed, and in a cold sweat. The blood pressure, formerly 155/90, was now 110/80. He had auricular fibrillation with a ventricular rate of 150, but his appearance was very different from that which he presented during former and subsequent paroxysms of the same arrhythmia. An electrocardiogram showed ventricular complexes like those in Fig. 1*B*, except that the Q_3 which had been present before was still there.* The next day the fibrillation stopped; the RS-T interval deviations diminished in size, but they did not disappear until April 25, 1933. For a month the contour of the electrocardiogram kept changing. No fever or leucocytosis occurred. The patient recovered and was discharged May 26, 1933. The third attack of cardiac pain was initiated by strenuous effort on June 26, 1934, and lasted until death on June 30, 1934. During this final episode, auricular fibrillation came and went. Marked RS-T interval deviations appeared in the electrocardiogram, and the blood pressure dropped to 80/60. This may have been an example of cardiac infarction without recent coronary occlusion.

Digitalis was never given to this patient except (a) from July 28 to Aug. 3, 1932, when he received 21 grains of powdered leaf, and (b) on June 30, 1934, when he received 24 c.c. of the tincture.

CASE 11.—F. K. was a mildly diabetic white man 58 years old. On Jan. 23, 1934, he had a severe substernal pain; then he developed cough, fever, leucocytosis, tachycardia, basal râles, and distant heart sounds. He was admitted to the Philadelphia General Hospital Jan 26, 1934, on the service of Dr. Dillon. At first there was some doubt whether the patient had coronary occlusion or a pulmonary infection. However, recurring severe attacks of substernal pain finally convinced the attending physician that the diagnosis of coronary occlusion was correct. Many electrocardiograms (22 in all) were taken. The one here reproduced (Fig. 3*B*) was obtained Feb. 3, 1934, two days after the most severe attack of all. Left axis deviation was present. The patient gradually improved. RS-T interval deviations had subsided markedly by Feb. 16, 1934. On Feb. 24, 1934, they had disappeared completely, leaving a tracing with T_1 and T_2 diphasic, T_3 and T_4 normal, and left axis

*The electrocardiograms in a similar case have been published.[4] The patient had a significant Q_3 before and after a possible lateral infarction.

deviation. The patient was discharged April 3, 1934. At present he has angina of effort. No digitalis was administered at any time.

CASE 12.—J. McH. was a white man 68 years old who developed severe substernal pain Sept. 21, 1931, which was accompanied and followed by all the clinical signs of coronary thrombosis. On Sept. 28, 1931, his physician, Dr. Henry Wise, asked us to take an electrocardiogram. The tracing has certain resemblances to that in Case 1, with, in addition, left axis deviation. The patient recovered and returned to work on a limited schedule on Oct. 3, 1932. On March 5, 1938, he was somewhat incapacitated as a result of two attacks of cerebral thrombosis in 1937. His heart has "given him no further trouble." No digitalis was given before the tracing was taken.

CASE 13.—M. C., a white man of 62, had an attack of coronary occlusion Nov. 1, 1932, and a recurrence of pain Dec. 5, 1932. Digitalis, in doses of 10 drops t.i.d., was begun after the second attack. An electrocardiogram which was taken Dec. 19, 1932, resembled Fig. 1*B*. Left axis deviation was also present. Digitalis was stopped; recovery took place, and Feb. 9, 1933, another tracing showed almost complete disappearance of RS-T interval deviations. He is reported to have died Dec. 10, 1934, as a result of his "heart lesion complicated by a kidney and bladder infection."

CASE 14.—T. C., a white man aged 50 years, had suffered with coronary symptoms for two years. In November, 1935, a marked exacerbation occurred. An electrocardiogram made Dec. 6, 1935, showed changes resembling those in Fig. 1*B*. On Dec. 9, 1935, he was referred to the Medical Ward of the Philadelphia General Hospital, on the service of Dr. Schnabel, with the diagnosis of "hypertension, angina, and possible coronary occlusion." There was a marked difference between the blood pressure in the two arms, but no signs of aortic aneurysm were elicited. By December 11 RS-T interval deviations had subsided considerably. In a tracing taken December 31, they were absent. T_1, T_2, and T_4 were inverted. Slight left axis deviation was present. He recovered and was discharged Jan. 4, 1936. No digitalis was administered.

CASE 15.—M. F. was a white man 74 years old who had a cystotomy for acute retention Sept. 4, 1935, in the Philadelphia General Hospital, on the service of Dr. Jump. After operation he complained of precordial pain, tachycardia developed, and the blood pressure changed from 130/80 to 100/80. He grew weaker and died Oct. 7, 1935. Digitalis was administered from Aug. 23, 1935, to Sept. 23, 1935; from August 23 to 29, the dose was 4 grains daily; from August 29 to September 4, 2 grains daily; and thereafter 3 grains daily. The only electrocardiogram was taken Sept. 19, 1935. It resembles Fig. 1*B* with left axis deviation in addition. The report of this tracing states that it was not possible to tell whether the RS-T interval deviations were due to coronary occlusion or to digitalis. The cause of this patient's death was apparently not clearly understood at the time. Review of the history and findings makes us feel that he probably had a lateral infarction shortly after operation.

CASE 16.—J. Y., a white man aged 59 years who was known to have hypertension, suffered an attack of pain in the chest and epigastrium, accompanied by dyspnea, on Jan. 9, 1936. He was admitted to the Philadelphia General Hospital on the service of Dr. Schnabel on Jan. 11, 1936, with tachycardia and slight fever. The blood pressure did not drop. An electrocardiogram which looks like Fig. 1*B* was taken January 13. Digitalis in doses of 2 c.c. daily was begun January 15. On January 17 the RS-T interval deviations had decreased in amplitude. Digitalis

was increased to 3 c.c. daily on March 3, 1936, and to 4 c.c. daily on March 12. On March 15 it was reduced to 3 c.c. daily and continued at this dose until discharge. The third tracing was taken March 16, 1936, when, on clinical grounds, the patient's lesion was thought to be quite well healed. In this electrocardiogram, however, the typical pattern of acute lateral infarction is seen, with RS-T interval deviations which are more marked than those on Jan. 17, 1936. The patient was discharged March 27, 1936, with a markedly reduced exercise tolerance. Later, he went into congestive failure and died Feb. 10, 1937.

CASE 17.—M. G., a white man 53 years old, had been rejected for insurance at the age of 41 because of a heart murmur, but except for a little dyspnea on effort he had been subjectively well until June 8, 1933. That afternoon, while at work, he had a severe attack of substernal pain, experienced rapid, irregular palpitation, became weak and short of breath, and thought he would die. He lay down for two hours. When he tried to get up, constriction over the heart and dyspnea occurred. Subsequently the systolic blood pressure fell 25 mm. and slight fever appeared. "Small doses" of digitalis were given for the next three days. An electrocardiogram (Fig. 7*A*), which was taken June 12, 1933, showed auricular fibrillation with ventricular complexes like those of Fig. 1*B*. He was admitted to the Philadelphia General Hospital June 12, 1933, on the service of Dr. Robertson. Between June 12 and 17 11 c.c. of tincture of digitalis were given. On the latter date the tracing (Fig. 7*B*) showed a slower ventricular rate and a disappearance of RS-T interval deviations. Digitalis was continued in doses of 4 c.c. of the tincture daily from June 18 to 24, and 3 cat units of powdered leaf daily from July 2 to August 14. The patient showed no toxic symptoms. He improved gradually and was discharged Aug. 14, 1933. On Aug. 10, 1933, clinical evidence suggested that the infarct had healed. Nevertheless, an electrocardiogram on that day (Fig. 7*C*) showed the most marked RS-T interval deviations of all. The typical pattern of acute lateral infarction was present. After discharge the patient led a restricted life with a markedly reduced exercise tolerance. Three years later he had another severe attack of cardiac pain and developed signs of congestive heart failure. Slow improvement took place for two weeks. Then, on Aug. 13, 1936, sudden death occurred with symptoms suggesting cerebral embolism. There was no necropsy.

There was a great deal of discussion as to whether the patient had an attack of coronary occlusion June 8, 1933, or merely a paroxysm of auricular fibrillation. The clinicians in charge were convinced that a coronary occlusion had occurred. The electrocardiographer was uncertain because the RS-T interval deviations "resembled digitalis effects" and because they were reproduced later by digitalis administration. Unless this patient actually had a coronary thrombosis June 8, 1933, it is difficult to explain the marked permanent reduction of exercise tolerance, the very slow recovery, or the reduction in size of the RS-T interval deviations between June 12 and 17, 1933, when the dose of digitalis was increased.

CASE 18.—I. L., an elderly white man, began to suffer recurring attacks of pain in the left chest about the middle of March, 1935. Some were referred to the right shoulder and arm, and lasted an hour. He was admitted to the Philadelphia General Hospital on the service of Dr. English, April 29, 1935, with epigastric pain, a rapid irregular pulse, slight fever, and a blood pressure of 150/70. The patient subsequently became psychotic. On May 14, 1935, he spent a violent night. The next day he was cyanosed and in shock with a blood pressure of 50 systolic and a heart rate of 30. On May 16, 1935, he died suddenly. There was no necropsy.

From April 29 to May 3, 1935, 4 c.c. of tincture of digitalis were given daily. On the latter date an electrocardiogram was taken which showed auricular fibrillation and ventricular complexes like those in Fig. 1*B*. On May 6 the interne made a

note that he had expected the electrocardiogram to show evidences of coronary occlusion, whereas the report showed only auricular fibrillation and digitalis effects. Tracings taken May 14 and 15, after his collapse, showed normal rhythm and signs suggesting infarction in the posterior surface of the left ventricle.

CASE 19.—G. E., an Armenian male of 54 years, had complained of increasing dyspnea and palpitation for the preceding year. On March 16, 1938, he was admitted to the University of Pennsylvania Hospital on account of lower abdominal pain. After considerable study, a tentative diagnosis of a small mesenteric thrombosis was made. At 10 A.M. on March 25, 1938, a severe epigastric and substernal pain began, and the blood pressure fell from 205/130 to 90/70. The pulse became weak and showed an extrasystolic arrhythmia. The patient became cold, weak, cyanotic, dyspneic, anxious, and sweaty. During the next few days there were slight fever and leucocytosis. The blood pressure slowly regained its former level over a period of three days. The acute episode subsided and the patient signed his release on March 31, 1938.

An electrocardiogram made March 25 (Fig. 2*B*), four hours after the onset of the attack, showed signs suggesting acute lateral infarction with left axis deviation. By the next day (Fig. 2*C*) the evidences of acute infarction had disappeared. No digitalis was administered to this patient.

The rapid disappearance of the evidences of acute myocardial ischemia in this case suggests that considerable constriction of collateral vessels may have accompanied the original coronary occlusion. There may have been a large area of temporary ischemia on March 25 and only a small area of permanent damage on March 26.

CASE 20.—B. C., a white man of 52 years, had an attack of severe substernal pain on March 6, 1938, which radiated to the neck. After he received ½ gr. of morphine, his heart "stopped beating for a minute," and the physician thought the patient was dead, but he recovered. On March 7 slight fever appeared. On March 9 another severe attack of pain occurred, and he was admitted to the Philadelphia General Hospital, on the service of Dr. Schaeffer, with tachycardia, a blood pressure of 100/80, cyanosis, basal râles, leucocytosis (12,000), and no fever. Another pain occurred March 16. Thereafter he improved slowly but steadily. He received no digitalis at any time. A number of electrocardiograms were taken. On admission the ventricular complexes resembled those in Fig. 1*B* except that left axis deviation was present, and the RS-T interval deviations in limb leads were less marked. By March 16 RS-T interval deviations had disappeared, T_1 and T_4 were inverted. On March 30 the electrocardiogram was entirely normal except for left axis deviation.

CASE 21.—M. N. was a white man of 33 who, in November, 1933, began to have attacks of burning pain in the epigastrium and substernal region, produced by effort and relieved by rest. These continued until his admission to the University of Pennsylvania Hospital May 15, 1934. On account of his youth, the diagnosis of angina was scarcely credited by the physicians in charge and the patient was sent to the electrocardiographic room May 19, 1934, with a request that tracings be taken before and after exercise. The control tracing taken at 4:05 P.M. (Fig. 5*A*) is normal. The patient then induced lower sternal burning discomfort of moderate degree by stepping up on a chair twenty-five times and then swinging the arms a few times. During this discomfort, at 4:15 P.M., the electrocardiogram shown in Fig. 5*B* was taken, and it shows the typical pattern of lateral infarction. By 4:29 P.M. the discomfort had subsided completely. At 4:35 P.M. the tracing had returned to normal (Fig. 5*C*). There never was any suggestion that this episode was anything but an attack of effort angina. Electrocardiograms taken subsequently, on May 23

and 31, 1934, and July 12, 1934, were all within normal limits. The patient is reported to have dropped dead in a bus in August, 1934. Similar electrocardiograms obtained during transient myocardial ischemia have been reported by Jervel[5a] and by Levy, Barach, and Bruenn.[5b]

CASE 22.—R. Z. was a woman of 42 years who had had diabetes eighteen years, since 1920. She began to suffer with intermittent claudication in 1930. In 1935 dyspnea and palpitation on effort appeared, and hypertension was discovered. In November, 1936, the patient had a few fainting spells. On March 1, 1938, mild precordial discomfort appeared on effort. On March 25 she was brought to the University of Pennsylvania Hospital in an acute attack of coronary occlusion, which probably began early that morning. She was cold and clammy. The blood pressure was 110/70. The pulse rate was 104 per minute. The next day she was a little better; the leucocyte count was 23,000, and there were râles in the lungs, especially on the left side. On March 27 she had a paroxysm of auricular fibrillation which lasted from 10 A.M. to noon, and the blood pressure dropped to 75/55. That night she had several attacks of severe dyspnea, and became stuporous. On the morning of March 28 she was again found to have auricular fibrillation. At 9:45 A.M. an attack of pulmonary edema brought about her death within fifteen minutes.

Necropsy was performed by Dr. Lippincott on March 28, 1938. The heart was of normal size. The right coronary artery was sclerosed but patent. The left anterior descending artery was occluded by organized calcified tissue. The circumflex artery was markedly narrowed 1 cm. from its origin by chronic atherosclerosis. At this point a recent red thrombus occluded the remainder of the lumen. An extensive area of infarction involved the upper half of the anterior surface of the left ventricle and the entire lateral wall, extending around well into the posterolateral region. It did not quite reach the interventricular septum posteriorly. The posterolateral portion of the infarction involved the inner half of the ventricular wall, leaving the epicardial surface relatively undamaged.

Electrocardiograms were taken March 25 and 26, 1938. On the first day (Fig. 6*A*) the limb leads showed a pattern suggesting acute anterior infarction. The precordial leads, however, were unusual: CF_3 showed an RS-T interval depression, suggesting posterior or lateral infarction; CF_4 showed a normal ventricular complex; CF_5 (in which the exploring electrode must have been considerably to the left of the apex, since the heart was not enlarged) showed a Q-wave and a marked RS-T interval elevation. On the following day (Fig. 6*B*) the pattern in the limb leads, though slightly different, was even more definitely that of anterior infarction. The precordial leads had changed markedly: CF_3 and CF_4 now showed a deep Q-wave and an elevated RS-T interval; CF_5 showed a much greater RS-T interval deviation than CF_3 or CF_4. Thus in neither of the tracings were the findings in precordial leads those of typical anterior infarction. On the first day the diagnosis of anterolateral infarction was made on the basis of the electrocardiogram. On March 26 the changes which had occurred since the day before suggested to us the possibility that the lesion had extended further forward into the anterior wall of the left ventricle; and that by dominating the RS-T interval deviation in CF_3 it had obscured the evidences of lateral infarction in this lead which had been seen on the previous day.

CASE 23.—J. S., a white man 59 years old, had hypertension and arteriosclerosis. On Jan. 10, 1938, he began to have epigastric pain, anorexia, cough, expectoration, dyspnea, orthopnea, and attacks of palpitation. He was admitted to the Philadelphia General Hospital on the service of Dr. Thomas Klein, on Jan. 18, 1938, looking very ill. The blood pressure had fallen from an earlier level of 200/100 to 105/75; the leucocyte count was 18,600; there were evidences of congestive heart failure. On Jan. 24, 1938, death occurred during a recurrence of cardiac pain.

A necropsy was performed that day by Dr. Ehrich. The heart was moderately enlarged. The left anterior descending artery was occluded 1 cm. from its origin by an old thrombus. The right coronary artery was very small. The left circumflex, a very large vessel, which continued on to form the posterior descending artery, was occluded 2 cm. from its origin by a recent thrombus. There was acute necrosis of the entire posterior and lateral wall of the left ventricle and the posterior third of the interventricular septum. The infarct extended from apex to base and from ventricular septum to the anterolateral portion of the ventricle—a very large lesion. In addition there was a large healed infarct in the anterior surface of the left ventricle, involving also the anterior part of the interventricular septum.

Electrocardiograms were taken Jan. 19, 20, and 21, 1938. The first shows a peculiar type of tachycardia, probably ventricular. The second (Fig. 6*D*) and third show a pattern much like that in Case 1, except that in Lead III there are a Q-wave, an elevated RS-T interval, and an inverted T-wave. The tracing shows a combination of the electrocardiographic signs of posterior and lateral infarction. The patient received 4 grains of powdered digitalis on Jan. 18, 1938.

CASE 24.—N. G. was a man of 69 years, a patient of Dr. P. Boland Hughes and Dr. Kendall A. Elsom. We are indebted to them for the privilege of reporting his findings.

On Dec. 17, 1937, the patient experienced a severe epigastric pain which radiated to the lower abdomen. It lasted from two to three hours, made him sweat, and caused him to feel distended. After the pain left, a soreness in the epigastrium persisted, and he felt short of breath, even when lying still. Examination showed slight cyanosis, a few basal râles, and a blood pressure of 120/80. On Dec. 23, 1937, there was a recurrence of epigastric pain and dyspnea. That day a transient but very definite pericardial friction rub was heard at the apex. On Dec. 26, 1937, urinary retention occurred. On Dec. 28, 1937, a left-sided pulmonary lesion was suspected. On Jan. 3, 1938, he seemed to be improving. That morning at eleven o'clock he had a sudden attack of dyspnea, cyanosis, and sweating. The pulse became imperceptible. At 4:30 P.M. a similar attack occurred which proved fatal.

A necropsy was performed by Dr. Lippincott on the day of death. The heart was not enlarged. The left anterior descending coronary artery was obstructed 3 cm. from its origin by calcified tissue. The right coronary artery was completely occluded 3 cm. from its origin by an old thrombus. The left circumflex artery was a very large vessel. In an upper branch an old calcified obstruction was found. The main vessel was blocked 2 cm. from its origin by a recent red thrombus. There was a large infarct involving the posterior and lateral surfaces of the left ventricle. The septum was scarred but had no fresh infarction. The anterior surface of the left ventricle showed a small scar but no recent muscle damage.

Electrocardiograms were taken four times. On Dec. 20, 1937, the tracing was normal. On Dec. 23, 1937, the RS-T interval was slightly elevated in Leads II and III and depressed in Lead IV. On December 24 these deviations were less marked. On Dec. 29, 1937, the last tracing was taken; it showed definite RS-T interval deviations in Leads II, III, and IV (Fig. 6*C*). No digitalis was administered at any time.

CASE 25.—C. D., a white man of 49 years, had been drinking heavily for two years and had developed psychotic tendencies. On Nov. 11, 1937, while walking on the street, he experienced a severe substernal pain which radiated to the left arm and epigastrium. Vomiting, dyspnea, and palpitation occurred. He was taken home and given morphine. Subsequently cough and hemoptysis developed. He was admitted to the Philadelphia General Hospital on the service of Dr. Burr Nov. 17, 1937, cyanosed, dyspneic, and psychotic. The blood pressure was 110/65. The heart was totally irregular. Several physicians who saw him agreed that the

arrhythmia was due to auricular fibrillation. A loud apical systolic murmur and a few basal râles were heard. The next day the rhythm of the heart became regular, and the patient's general condition improved somewhat. On Nov. 21, 1937, at 7:30 A.M., a severe attack of substernal pain occurred. The patient became dyspneic, the lungs congested, and the pulse weak and rapid; the blood pressure fell to 75/50. The patient rallied again. On Nov. 26, 1937, cardiac arrhythmia reappeared. On Nov. 30, 1937, the patient died, presumably from "rheumatic heart disease."

Necropsy was performed by Dr. McCutcheon on the day of death. A "buttonhole" mitral stenosis was found. The entire pericardium showed fresh pericarditis. The right coronary artery was small but patent. The left anterior descending artery was of normal size and patent. The left circumflex, a very large vessel which continued on to form the posterior descending artery, was occluded 1 cm. from its origin by a red thrombus. A large recent infarct involved the entire posterior surface of the left ventricle.

Electrocardiograms were taken Nov. 18 and 23, 1937. The limb leads showed right axis deviation, notched P-waves, and upright T-waves. The patient received 8 c.c. of digalen intramuscularly on Nov. 22, 1937, 2 c.c. on Nov. 23, 1937, and a daily dose of 1½ grains of powdered leaf thereafter. It is surprising that the only electrocardiographic evidence of this large infarct was the depression of the RS-T interval in Lead IV.

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THE HEART IN PNEUMOCONIOSIS*

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ALTHOUGH pneumoconiosis has received much attention in recent literature as the most important industrial disease of today,[1] relatively little has been said concerning the associated cardiac change, namely, cor pulmonale (pulmonary heart disease).

A brief review of the pathology of pneumoconiosis will explain why cor pulmonale occurs. The pneumoconioses (*pneumon*—lung; *konis*—dust) are caused by the inhalation of minute particles of organic or inorganic dust. In recent years it has become recognized that probably the only substance producing changes of clinical significance is free silica (SiO_2). Most of the silicates are harmless except asbestos (magnesium, calcium silicate), which produces asbestosis. Coal dust and metallic dust seem to be of importance only if combined with free silica.[2] The particles of silica are phagocytized and carried into the lymph channels, which become obstructed. Silica stimulates fibrosis. The fibrous tissue forms around the bronchi and small branches of the pulmonary arteries, and gradually replaces the lung tissue. Pneumoconiosis has been classified into three stages, according to the degree of fibrosis. Recently there is a tendency to substitute a more detailed pathologic roentgenologic classification.[3, 4]

Jaffe[2] stated: "The replacement of large parts of the lungs by a very poorly vascularized scar tissue and obliterating changes in the branches of the pulmonary artery interfere greatly with the pulmonary circulation. The increase in resistance to the blood flow causes hypertrophy of the right ventricle of the heart, the wall of which becomes thickened to from 6 to 10 mm. as compared with a normal thickness of from 3 to 4 mm. With the exhaustion of the reserve power the hypertrophic ventricle becomes decompensated and failure of the right heart is a common cause of death in advanced pulmonary silicosis."

With this in mind, we undertook to study the incidence of pulmonary heart disease in pneumoconiosis and its frequency as a cause of congestive failure. Second, we wished to ascertain whether the cardiac changes could be detected clinically and roentgenologically.

MATERIAL

Our material consisted of 205 cases of pneumoconiosis from the Los Angeles County Hospital. Of the 19,800 patients who came to necropsy

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in the last twenty years, 102 had pneumoconiosis. We included as pneumoconiosis cases of silicosis, anthracosis with moderate or marked fibrosis (anthracosilicosis), and pulmonary fibrosis in which the etiology was unknown provided the silica content of the wet lung tissue was more than 2.5 mg. per cent.[5*] There were also 103 other patients who had both an adequate history of exposure to silica and radiographs which showed the characteristic pulmonary changes of pneumoconiosis. In four of the patients who came to autopsy the clinical records were missing. We reviewed the roentgenograms in 105 cases in which they were still available.

ANALYSIS OF CLINICAL DATA

In the 201 cases in which histories were available, the disease was advanced; 55 were classified as moderately advanced (second stage), and 146 as far advanced (third stage). Most of the patients had been miners for a long period of years.

We found that pneumoconiosis is a disease of men past 40; in 31.5 per cent of the cases the patients were over 60 years of age, in 66 per cent over 50 years of age, and in only 8.4 per cent below 40 years of age. Cases of acute silicosis have been reported in young individuals after short periods of exposure.[6] One of our patients was less than 30 years of age, but he also had pulmonary tuberculosis.

Dyspnea was mentioned in 142 cases but could not be used as an index of cardiac involvement because it is also one of the cardinal symptoms of advanced pneumoconiosis. In 55.6 per cent of these cases dyspnea had been present for more than a year; in 27.5 per cent it had been noticed for over five years; and in one case had been present as long as thirty years. Cough was usually associated with the dyspnea and had had approximately the same duration.

On physical examination cardiac enlargement, as shown by palpation and percussion, was thought to be present in 23.8 per cent of the 201 cases. Auscultatory examination revealed that the heart sounds were distant in fifty-four instances (26.3 per cent). Cardiac murmurs occurred in twenty cases in which organic heart disease, other than cor pulmonale, was excluded.

Cor pulmonale is one of the types of heart disease in which the cardiac rhythm tends to remain normal throughout.[7] In this series auricular fibrillation occurred in six cases of pneumoconiosis in which cor pulmonale was apparently the only heart disease present. In two of these cases this was confirmed by electrocardiographic and autopsy evidence, respectively.

We believe that accentuation of the pulmonic second sound and the presence of marked cyanosis are suggestive of cardiac involvement in

*The microdeterminations were kindly done by Albert L. Chaney, Ph.D., chemist at the Los Angeles County Hospital.

pneumoconiosis. There was other evidence of cardiac failure in 63 per cent of the twenty-two cases in which the pulmonic second sound was accentuated. In 88 per cent of the twenty-seven cases in which there was marked cyanosis, other signs of cardiac failure were also present.

Serologic tests for syphilis were recorded in 166 cases, in 19.8 per cent of which the reaction was positive. This is approximately three times the general incidence of syphilis in the hospital population.

Diseases interfering with the pulmonary circulation are frequently thought to cause compensatory polycythemia. This was not substantiated by the findings in our series. The hemoglobin content and the erythrocytes were rarely increased, and not infrequently anemia was present. The hemoglobin content had been measured in seventy-two cases, and in only two (2.7 per cent) was it above 100 per cent. Some degree of anemia (hemoglobin below 80 per cent) was present in 45.8 per cent. The erythrocytes had been counted in seventy cases and were found to number more than 5,500,000 per cubic millimeter in only three instances (4.3 per cent), whereas in 18.5 per cent of the cases the count was below 4,000,000.

Electrocardiograms were available in 43 cases, in 20 of which an autopsy had been performed. Of these 43 cases, the axis deviation was right in 16 (37.2 per cent), normal in 19, and left in 8.

Right axis deviation closely paralleled the autopsy evidence of right ventricular hypertrophy. There were 10 autopsy cases in which right axis deviation had been present. Of these, pure right ventricular hypertrophy was found in 8, marked dilatation of the right heart without hypertrophy in one, and hypertrophy of both ventricles in one. In only one case in which there had been no abnormal deviation of the electrical axis was right ventricular hypertrophy found at autopsy, and in this instance pericarditis was also present.

The electrocardiograms in 24 cases of pneumoconiosis in which there was no known organic heart disease other than cor pulmonale showed evidence of myocardial damage in 9 (low voltage in 5 cases and inversion of T_2 and T_3 in 4 cases). Two other tracings showed auricular fibrillation.

CONGESTIVE FAILURE

The term "definite congestive failure" was reserved for patients who came to necropsy with marked chronic passive congestion of the liver and either ascites or edema, for cases in which the pathologist made a diagnosis of "congestive failure," and for living patients whose ascites or edema led to the clinical impression of "congestive failure." All patients in whom the evidence of congestive failure was less convincing, but suggestive, were classified as instances of "questionable failure."

We found that congestive failure occurs especially in the third stage of pneumoconiosis and is a rather common cause of death. To

determine the incidence of failure we divided the patients into two groups: (1) those living, according to last available records, and (2) those whose records showed that they died. There were eighty-four living patients in the total group, 10.7 per cent of whom had definite congestive failure. There were 121 patients who died, 48.8 per cent of whom had had definite congestive failure (Table I A). We also divided patients who had no evidence of cardiac involvement except cor pulmonale from those who had hypertension (blood pressure 150/100 or more) or organic heart disease other than pulmonary heart disease. Nine per cent of the sixty-seven living patients without heart disease other than cor pulmonale had definite congestive failure. Most of these were in the third stage of pneumoconiosis. Forty-seven and two-tenths per cent of the eighty-nine patients who died had had definite congestive failure. Nearly 80 per cent of these were cases of third stage pneumoconiosis (Table I B).

TABLE I

A. INCIDENCE OF CONGESTIVE FAILURE IN 205 CASES OF PNEUMOCONIOSIS

LIVING PATIENTS (TOTAL, 84)		DECEASED PATIENTS (TOTAL, 121)	
QUESTIONABLE FAILURE	DEFINITE FAILURE	QUESTIONABLE FAILURE	DEFINITE FAILURE
10.7%	10.7%	14.0%	48.8%

B. INCIDENCE OF CONGESTIVE FAILURE IN 156 CASES OF PNEUMOCONIOSIS WITHOUT HEART DISEASE EXCEPT COR PULMONALE

	LIVING PATIENTS (TOTAL, 67)		DECEASED PATIENTS (TOTAL, 89)	
	QUESTIONABLE	DEFINITE	QUESTIONABLE	DEFINITE
Stage II	4.5%	1.5%	2.3%	10.1%
Stage III	4.5%	7.5%	10.1%	37.1%
Total	9.0%	9.0%	12.4%	47.2%

The late occurrence of congestive failure in pneumoconiosis was further indicated by a study of the duration of the signs of congestive failure. In the group of fifty-three patients without heart disease other than cor pulmonale who died with congestive failure, its duration had been over two months in only two, three months in one, and seven months in the other. This evidence seems quite conclusive that cardiac failure is a terminal event and that when it occurs the prognosis is grave.

Our findings are not in agreement with the popular concept that 75 per cent or more of silicosis patients die of pulmonary tuberculosis.[8, 12] Definite congestive heart failure occurred more frequently in our patients than did tuberculosis. The sputum was examined in 103 cases and tubercle bacilli were found in forty-two (40.8 per cent).

In the autopsy material 41 of the 102 patients (40.2 per cent) had had tuberculosis, while 52 (51.0 per cent) had had definite congestive failure.

It has been thought that patients with anthracosilicosis are less susceptible to tuberculosis than patients with pure silicosis.[9] The comparatively lower incidence of tuberculosis in our series may be partially explained by the fact that some cases of anthracosilicosis were included.

RADIOGRAPHIC FINDINGS

We reviewed the radiographs which were available in 105 of our cases. In twenty-seven of these cases the lesion was verified at autopsy. An effort was made to determine the number in which cardiac enlargement was present and to learn whether or not we could confirm reports of constant changes in the posteroanterior (sagittal) cardiac shadow associated with right ventricular enlargement.

The right ventricle lies almost entirely on the anterior and diaphragmatic surfaces of the heart and does not of itself form any material part of the cardiac silhouette in the posteroanterior view. We recognize the value of lateral and oblique views in detecting right ventricular enlargement, as demonstrated by Fray,[10] Pancoast and Pendergrass,[3] Parkinson and Hoyle,[11] and others. Our material consists entirely of routine sagittal films, and therefore we are limiting our discussion to the findings in these.

The films were first reviewed independently. The findings were then compared with the clinical and pathologic data. The points covered were: (1) cardiac enlargement by inspection; (2) prominence of the pulmonary conus; (3) elevation of the apex; (4) prominence of pulmonary vascular shadows; (5) enlargement of the right auricle; and (6) cardiac measurements in longitudinal, basal, and transverse diameters, and the cardiothoracic ratio.

Enlargement by Inspection.—In each case the heart was classified as to its size and its type of enlargement according to the general impression gained from inspection of the films only. The pulmonary changes so obscured the heart shadow that in many cases the impression of cardiac size was unreliable, and in 4.7 per cent of the entire group absolutely no impression of cardiac size could be obtained.

Pancoast and Pendergrass[3] state: "It is a striking feature that advanced silicotics who have developed mild myocardial degeneration do not, as a rule, present the roentgenologic evidences of a large cardiac shadow in the sagittal view, but it may be demonstrated in the lateral direction." The overexpansion of the lung in silicosis makes the sagittal cardiac silhouette less reliable as a measure of cardiac size because of the increase in posteroanterior chest diameter and low position of the diaphragm. Lanza,[12] while admitting the theoretical basis for right heart hypertrophy, states that in pneumo-

coniosis large heart shadows are not seen in the films and that the heart often appears small and "hanging."

In 78 of the 105 cases in which radiographs were available the patients had no heart disease other than cor pulmonale. The findings in this group support the above statements. Right ventricular hypertrophy did not usually cause characteristic enlargement of the posteroanterior cardiac silhouette. The heart appeared definitely enlarged in 24.3 per cent of these 78 cases, but in only 5.1 per cent was the enlargement thought to be right ventricular.

From a study of the films in 27 autopsy cases, we found that all but one of 11 hearts weighing over 400 gm. were represented by recognizably enlarged silhouettes. Right ventricular hypertrophy was rarely determined by inspection of the routine posteroanterior films. Only 2 of 10 autopsy cases with right ventricular hypertrophy were identified as such by inspection of the sagittal films alone. The hearts in 5 of the 10 cases were considered radiographically normal; 4 of these hearts weighed 380 gm. or less.

These facts support our conclusions and the statements of others that right ventricular hypertrophy may be present without enlargement of the posteroanterior cardiac silhouette.

Prominence of Pulmonary Conus.—Enlargement of the pulmonary conus, the outflow channel of the right ventricle, has been mentioned as radiographic evidence of right ventricular enlargement. In a series of 127 cases of third-stage or advanced second-stage anthracosilicosis, Dyson[13] found enlargement of the pulmonary conus in 14 per cent and attributed it to right ventricular enlargement. In his single autopsy case this idea was confirmed. Numerous authors have pointed out the association of enlargement of the pulmonary conus with right ventricular hypertrophy in congenital and in mitral heart disease.

Our findings indicate that enlargement of the pulmonary conus, when present in pneumoconiosis, usually accompanies definite right ventricular hypertrophy. The pulmonary conus was seen to be definitely enlarged in approximately 22 per cent of the entire group of 105 cases. In 28.5 per cent this area was obscured by overlying pulmonary markings. In eleven cases with autopsy proof of right ventricular enlargement (in which films were available), the pulmonary conus was obscured in two. In five of the remaining nine cases the conus was definitely dilated. In one patient who came to autopsy the film showed dilatation of the conus and general enlargement of the heart, but the autopsy record failed to mention any abnormality in the heart.

Elevation of Apex.—Elevation of the cardiac apex should occur when the right ventricle, which constitutes the anterior and lower part of the heart, hypertrophies. In extreme cases this may lead to a typical *cor en sabot* such as is associated with certain congenital heart lesions.

However, Parkinson and Hoyle[11] failed to find this criterion of right ventricular hypertrophy in any of their cases of emphysema, and Dyson,[14] in his discussion of the roentgenologic appearance of the heart in anthracosilicosis, does not mention it. Our findings in this regard indicate that, although elevation of the apex is uncommonly seen in pneumoconiosis, when present it suggests right ventricular hypertrophy. In some cases it is difficult to determine the exact position of the apex without fluoroscopy.

Among the 27 cases of pneumoconiosis with autopsy records in which films were available, there were 10 with right ventricular hypertrophy. The apex was definitely elevated in 2, and slightly or questionably elevated in 3. It was also apparently elevated in one case with hypertrophy of both ventricles. In all autopsy cases in which the radiographs showed an elevation of the apex, the heart weighed at least 420 gm. and there was hypertrophy of the right or both ventricles.

Pulmonary Vascular Prominence.—Although Parkinson and Hoyle[11] found that prominence and tortuosity of the large branches of the pulmonary artery are the most frequent evidences of cardiac involvement in emphysema, we were unable to make use of this criterion in the radiographic study of pneumoconiosis because the pulmonary lesion blots out the vascular detail in most cases.

Enlargement of Right Auricle.—Our findings agree with those of Parkinson and Hoyle,[11] who logically regard enlargement of the right auricle as a late and rather unusual finding in cor pulmonale. This change, which alters the right cardiac border, appears therefore to be of little practical value.

*Measurements.**—The transverse diameter is one commonly used as a measure of cardiac size, but our findings, like those of Newcomer and Newcomer,[16] throw doubt on its value. Its value in pneumoconiosis is also questioned by Dyson,[14] who states that although practitioners in anthracite coal regions recognize cardiac failure as a frequent cause of death in pneumoconiosis, a definite lesion is not diagnosed clinically since there is no increase in the transverse cardiac diameter. Since the cardiothoracic ratio depends on the transverse diameter of the heart, it likewise is a poor criterion of cardiac enlargement. It was impossible to determine the various diameters in 12 to 18 per cent of the 105 cases (Table II).

Our findings lead us to believe that, of the diameters measured, the basal or broad diameter is the most sensitive index of right ventricular enlargement. This diameter was greater than the maximum normal in 47.5 per cent of the seventy-eight cases in which there was no cardiac disease other than cor pulmonale. This measurement was increased more often than any other, and the frequency of its increase

*Maximum and minimum normal figures for the various diameters were obtained from Levene and Reid.[15]

closely parallels the percentage of hearts with right ventricular enlargement found in the autopsy series of 102 cases. The cardiothoracic ratio, on the other hand, was above maximum normal in only 7.7 per cent of these seventy-eight cases (Table II).

The radiographic measurements in the group with heart disease other than cor pulmonale testified, as would be expected, to the presence of more advanced degrees of hypertrophy. Increased diameters, with the exception of the basal diameter, occurred more than twice as frequently in this group as in the group in which only cor pulmonale was present. The basal diameter was enlarged in only a slightly higher percentage in the former group of cases (59.2 per cent as compared to 47.5 per cent).

In the twenty-seven cases with both radiographic and autopsy records, the findings confirm the above observations. Although all cardiac diameters were above maximum normal in the large hearts (400 gm. or above), the cardiothoracic ratio was above maximum normal in only four of seven hearts which weighed over 500 gm. It therefore seems very unreliable as a criterion of cardiac enlargement. In the hearts weighing less than 400 gm. in which radiographic measurements were possible, all except the basal diameter were within normal limits. The basal diameter was increased in three of four cases of right ventricular hypertrophy in this group, and in only one case without right ventricular hypertrophy. The basal diameter, therefore, as previously stated, appeared to be the most sensitive index of right ventricular enlargement.

TABLE II

MEASUREMENTS OF THE HEART IN POSTEROANTERIOR ROENTGENOGRAMS OF 105 CASES OF PNEUMOCONIOSIS

	WITHOUT HEART DISEASE EXCEPT COR PULMONALE (%)	WITH OTHER CARDIAC DISEASE (%)
	(78 cases)	(27 cases)
Longitudinal		
Obscured	16.7	3.7
Above maximum normal (15.0 cm.)	25.6	59.3
Broad or basal		
Obscured	20.5	7.4
Above maximum normal (10.5 cm.)	47.5	59.3
Transverse		
Obscured	14.1	3.7
Above maximum normal (14.5 cm.)	11.5	51.8
Cardiothoracic ratio		
Obscured	14.1	3.7
Below minimum normal (1.92)	7.7	33.3

AUTOPSY FINDINGS

Among the 102 autopsy cases of pneumoconiosis, we found 29 with hypertension or organic heart disease other than cor pulmonale. They included 18 cases of hypertension, 5 of pericarditis, 3 of coronary

heart disease, 2 of rheumatic heart disease, and one case of thyrotoxicosis. This leaves 73 cases of pneumoconiosis without cardiac disease other than cor pulmonale.

Criteria of Hypertrophy.—Estimation of right or left ventricular hypertrophy is most accurately accomplished by weighing each ventricle separately.[17] Since only the cardiac weights and the mural thicknesses had been recorded by the autopsy surgeon, we were obliged to use the average thickness of the ventricular wall as a criterion of hypertrophy.

Hearts in which the right and left ventricular walls measured less than 4 mm. and not more than 12 mm. in average thickness, respectively, were considered normal. A right ventricular wall which averaged 5 mm., or a left ventricular wall which averaged over 15 mm. in thickness, was considered definitely hypertrophied. The term "hypertrophy of both ventricles" was used to designate hearts in which both right and left ventricles were definitely hypertrophied, and also hearts in which the wall of the right ventricle was 5-6 mm. and that of the left more than 12 mm. in thickness, or the wall of the right ventricle 6-7 mm. and that of the left more than 14 mm. Our criteria, therefore, restrict the term "right ventricular hypertrophy" to those hearts which have a *predominant* right ventricular hypertrophy. Although Thompson and White[18] found left ventricular strain to be the greatest cause of right ventricular hypertrophy, the great majority of their examples of right ventricular hypertrophy would have been listed, by our criteria, under "hypertrophy of both ventricles." We feel that in the group of cases without cardiac disease other than cor pulmonale the percentage of cases listed as "right ventricular hypertrophy" represents quite conservatively the percentage of cases of cor pulmonale caused by the pneumoconiosis. Our classification still leaves a small group of hearts with questionable hypertrophy of the right or the left ventricle which we classed as "questionably normal."

In some autopsy records, the thickness of the right ventricle or, rarely, of the left ventricle, was not stated. If not mentioned, we have considered the ventricle as normal. This added an unavoidable error, but one which would minimize and not overemphasize the effect of pneumoconiosis as a cause of right ventricular hypertrophy.

Ventricular Hypertrophy.—Exclusive right ventricular hypertrophy occurred in 44.1 per cent of the total of 102 autopsy cases of pneumoconiosis. Including those cases in which both ventricles were hypertrophied, the incidence of right ventricular hypertrophy was 58.8 per cent.

Among the seventy-three autopsy cases in which there was no heart disease other than cor pulmonale the incidence of exclusive hypertrophy of the right ventricle was 52.0 per cent; including those cases

in which both ventricles were hypertrophied, the incidence was 61.6 per cent (Table III). From these findings we feel justified in concluding that the right ventricle is hypertrophied either exclusively or together with the left ventricle in the majority of fatal cases of pneumoconiosis.

TABLE III

INCIDENCE OF VENTRICULAR HYPERTROPHY IN 102 AUTOPSY CASES OF PNEUMOCONIOSIS

	WITHOUT HEART DISEASE EXCEPT COR PULMONALE (%)	WITH OTHER CARDIAC DISEASE (%)	TOTAL (%)
	(73 cases)	(29 cases)	(102 cases)
Left	0.0	10.3	2.9
Normal	30.1	20.7	27.5
Normal (?)	8.2	17.2	10.8
Both	9.6	27.7	14.7
Right	52.1	24.1	44.1

Weight of Hearts.—The heart weight was increased in the majority of the cases of pneumoconiosis. The hearts in approximately two-thirds of the 102 cases weighed more than 350 gm. This is in accord with the findings of Sweany, Porsche and Douglass,[9] who found that in nearly all of their cases the heart weighed from 350 to 500 gm. and had a thick right ventricular wall. The very heavy hearts, those weighing above 500 gm., occurred more frequently in the patients with other associated cardiac disease than in those in which only cor pulmonale was present (Table IV).

TABLE IV

PERCENTAGE OF HEARTS IN VARIOUS WEIGHT GROUPS IN 102 AUTOPSY CASES OF PNEUMOCONIOSIS

HEART WEIGHT IN GRAMS	WITHOUT HEART DISEASE EXCEPT COR PULMONALE (%)	WITH OTHER CARDIAC DISEASE (%)	TOTAL (%)
	(73 cases)	(29 cases)	(102 cases)
300 or below	28.8	13.7	24.5
301-350	13.7	0.0	9.8
351-400	17.8	20.7	18.7
401-450	20.6	10.4	17.6
451-500	6.8	24.1	11.8
Above 500	12.3	31.1	17.6

SUMMARY AND CONCLUSIONS

1. Autopsy protocols of 102 cases of pneumoconiosis (occurring in nearly 20,000 autopsies), together with the clinical records of 103 additional cases in which there were adequate histories of exposure to silica and characteristic roentgenographic changes, were reviewed. This was done to determine the incidence of cor pulmonale (pulmonary heart disease) and congestive heart failure, and whether or not the cardiac changes could be detected clinically and roentgenologically.

2. At autopsy, right ventricular hypertrophy was found to occur in approximately one-half of the cases of pneumoconiosis. Exclusive right ventricular hypertrophy was present in 44.1 per cent of the total of 102 autopsy cases. Including hypertrophy of both ventricles, right ventricular hypertrophy occurred in 58.8 per cent of the cases.

3. Definite congestive heart failure was found more frequently in these cases (51 per cent) than was tuberculosis (40.2 per cent). It was usually a terminal event. Therefore, when it occurs clinically, the prognosis is grave. It may be concluded, furthermore, that if pneumoconiosis is uncomplicated by tuberculosis or other pulmonary infection death from cardiac failure is to be expected.

4. Our findings lead us to believe that the clinical diagnosis of cor pulmonale in pneumoconiosis is suggested by accentuation of the pulmonic second sound, marked cyanosis, right axis deviation in the electrocardiogram, and characteristic changes in the posteroanterior radiograph, namely, prominence of the pulmonary conus, elevation of the cardiac apex, and an increase of the broad or basal diameter, in the absence of enlargement of other diameters.

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THE SOCIAL COMPONENT IN HEART DISEASE*

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THE function of the heart as the core of the individual's life and strength has paramount significance for each human being. Damage to this essential life-maintaining organ constitutes a threat which varies in its menacing quality not only with the degree of severity of the disease, but with the individual emotional make-up. Potential sources of social difficulties exist in the very nature of heart disease itself: in the sudden danger of its acute phases, in the possibility of recurrence and progression, and in its chronicity. The incidence of heart disease in all age groups—in children, young adults, middle-aged and elderly persons—implies unfavorable influences in varying degrees upon all the activities of life.

In every cardiac service certain problems recur again and again; for example, there is the ambitious, capable adolescent removed from school and sport; the young adult wage earner in need of vocational rehabilitation and financial aid for dependents; the young widow with small children on marginal income; the individual permanently incapacitated for continued pursuit of a successful professional or industrial career. Many variations exist in these problems, even in cases in which the degree of heart damage and economic conditions are approximately the same. The points of difference among these patients lie frequently in the personal sphere, in the reaction of the patient to illness in general, to cardiac illness in particular, and in the neurotic gain the patient derives from the illness in relation to the various members of his family, his immediate associates, his work, and all of his other contacts. Equally significant factors in these problems often arise from the reaction of the various members of his family to the patient and his illness. Viewed from these angles, the treatment of heart disorders carries with it implications greater than mere medical therapy. Individualized evaluation of the patient in all of his significant relationships should be the focus of any effective plan of care.

The objectives for care of these patients will vary according to the stage of the illness at which recommendations for care are being made. Obviously, the primary aim for any particular patient will be restoration to a state of maximum well-being. Continuous thought will be given to prevention of recurrence, and retardation of progress, of the

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illness. When these can no longer be accomplished, efforts will be directed toward reduction of the individual's disabilities and palliation of the distress which results from chronic progressive disease. These objectives have their best chance of realization in clinics and hospitals where physicians and social workers function together with mutual understanding of immediate and ultimate aims and of their respective roles in assisting the patient to meet his problem.

This paper will approach the subject of the social component in heart disorders as they affect children, adolescents, and adults. The major emphasis in the consideration of the problems of children and adolescents will be on prevention. As to the problems of adults in whom the disease has already become established, attention will be focused principally on adjustments of the patient's life activities.

PROBLEMS OF CHILDREN AND ADOLESCENTS

Rheumatic heart disease is the most prevalent type of heart disease in children; it has been estimated that in this country about 80 per cent of all organic heart disease in children of school age is rheumatic.[1] Although the etiology of rheumatic fever is not yet known, environmental conditions of dampness, lack of sunlight, overcrowding, general poor physical hygiene, and inadequate nutrition seem to be contributory to its development. Of equal importance with the child's physical surroundings are the persons in immediate contact with him. During his early formative years the child is particularly susceptible to the emotional influences of his environment. Overanxiety of parents and relatives, deficiency in normal affectionate concern for the child, mismanagement in child training, family friction, or other tensions in the home may have an adverse psychologic effect of far-reaching consequence on the child's entire later life. The possibility or the advisability of care in the home during both the acute and chronic phases of his illness will be largely determined by all of these factors, so that they require thoughtful consideration on the part of physicians, nurses, and social workers responsible for the care of the patient.

A boy aged 14 years, convalescing from rheumatic fever, was discharged from a hospital to his home by a house officer on assurance by an apparently intelligent mother that she was able to carry out his recommendations for many months of complete bed rest.

The following week a social worker called to ascertain how the patient was adjusting himself. As she approached the suburban cottage, she heard gales of laughter and shouting. Passing by a low street-floor window, she saw the patient jumping about on his couch, surrounded by six or eight boys and an immense dog. The radio was on full blast. Just as she entered the house, the patient dashed out of bed to change the radio program and embrace the romping dog. No one else was at home.

The mother had been called to a hospital by the serious illness of her divorced husband, who was still dependent on her emotionally and financially.

While awaiting the mother's return, the worker observed great strips of wall paper separated from the wall because of dampness. The temperature of the house was so low as to chill the worker even in her heavy winter coat. Later it was explained that the melting snow had leaked into the cellar and had extinguished the furnace fire!

As the social worker studied the situation, she found a mother distraught by her former husband's illness, their continuing personal maladjustments despite divorce, the critical illness of a daughter in another hospital, the impending loss of their home, a long history of family friction, and the ever-present behavior problem of the patient, who had been for years impervious to any family effort at guidance.

Improvement under such conditions was unlikely following even the most ideal hospital care for rheumatic fever. This mother, though apparently intelligent and actually well intentioned, was neither emotionally, economically, nor physically equipped to meet this patient's needs. Considerable social case work, based on understanding of the emotional reactions of the members of this family and a knowledge of resources of the family and community, was required before this patient received adequate care away from home, with resulting arrest of his rheumatic disease and treatment of his personality difficulties. Complete bed rest with correct nutrition for a period of many months' duration in a peaceful, warm, dry, sunlit environment, with adequately supervised educational and recreational opportunities, was required for this patient. This is the optimum program for all patients. When it cannot be obtained or approximated in the home, a substitute home is recommended, either in an institution or adequate foster home, under expert medical, nursing and social direction.

Inactivity and separation from the daily school and play interests with friends lead inevitably to boredom and restlessness. Unhappy thrashing about in bed is not synonymous with complete rest.

The serious effects of loss of school work and lack of opportunities for development were exemplified in the case of a 22-year-old man admitted to a hospital for total thyroidectomy.[2] From the age of 8 years, rheumatic heart disease had deprived him of education, play, normal companionship, and regular occupation. Repeated exacerbations and a progressively increasing degree of cardiac involvement had finally brought him to a public infirmary with an extremely poor prognosis for life. His illness had gradually produced a taciturn, morose, ill-tempered youth with asocial tendencies. For a considerable time following his admission to the hospital for surgical treatment, all who were engaged in his care believed that they were dealing with a person of subnormal mentality. However, after total thyroidectomy, followed by clinical improvement, the conclusion was reached that his behavior and poor intellectual grasp were, in reality, the result of interrupted schooling and normal human relationships, general deprivation and lack of opportunity. An agreeable home, clothing, financial support, regular medical attention, re-education to life in a normal community away from hospitals and infirmaries, recreation, vocational guidance and placement, reassurance, and encouragement were all part of a social plan of treatment. As this treatment progressed, it yielded increasing evidence that the patient was an alert, intelligent, ambitious person, and, in the course of time, he made extraordinary progress in attaining an independent, contented, and self-maintaining existence.

The importance of a supervised plan of school studies, occupational therapy and recreation, gradually adjusted to the increasing capacity of the child, cannot be overemphasized. Some district nursing associations and women's organizations have furnished resources for occupational therapy and recreational facilities in the homes of children ill with heart disease. In Massachusetts the law[3] provides that the school committee of every town shall ascertain the number of physically handicapped children of school age unable to attend public school. The presence of five or more such children in any given town makes it obligatory for the committee to employ teachers for instruction in the individual homes or in institutions. If there are less than five children, the law is permissive. In certain instances in which children have had several years of institutional care for heart disease their continued school instruction has enabled them to meet the public school requirements for graduation. The admirable work for children with heart disease which has been undertaken in some states by the federal social security program could be extended with profit to other parts of the country.

Aftercare for children and adolescents with rheumatic heart disease is always a composite of adequate treatment and prevention. Proper attention to the first of these goals should result in the achievement of the other. Certain of the conclusions reached in a recent intensive study* of 1,000 children with rheumatic heart disease over a ten-year period are pertinent. From the point of view of prognosis, it is believed that the five or six years following the initial rheumatic infection are the most dangerous and difficult period. If patients pass through these years safely and do not develop moderate to severe rheumatic heart disease with cardiac enlargement, they will probably do well.† It is during this period that special emphasis must be put on medical supervision at regular intervals in order to detect the presence of rheumatic infection or the development of valvular damage. The most important single factor in aftercare is avoidance of infection, which needs repeated interpretation both to the patients and their families; patients are urged to isolate themselves from those who have colds, either at home or in the school. The importance of avoiding stair climbing no longer has the emphasis formerly placed upon it. If patients live on the second or third floor and walk up slowly, there will probably be no ill effects, provided they do not have active rheumatic fever. A warm climate, such as that of Florida, is not a panacea. The disease in some cases may subside faster in the South, but some of the children sent to Florida from the House of the

*These data were secured from Dr. T. Duckett Jones, Research Assistant, House of the Good Samaritan, Boston, an institution specializing in treatment and research of rheumatic heart disease, and are quoted with his permission.

†This takes into account that some of these patients with only slight heart damage may be among those who in their twenties or thirties will develop subacute bacterial endocarditis. There is no means of anticipating in which of these patients the disease will take this course.

Good Samaritan developed acute exacerbations of the disease while there. Special stress is placed on the importance of a well rounded diet, abundant in vitamins C and D, milk, and on adequate rest and the avoidance of severe fatigue.

As for occupation, with the exception of heavy laborious work there are no special restrictions for patients who have done well and developed little rheumatic heart disease. In other clinics, likewise, it has been observed that many patients have carried on normally in school, play, and in industry several years after a severe initial illness. However, it has been said that the largest proportion of all patients with rheumatic heart disease are left with conditions requiring some limitation of activity. In these cases there will be need for recreational and vocational guidance. For the girls the question of marriage and childbearing requires individualized advice.

During these years there are psychologic aspects which are of the greatest importance. A casual comment, in the child's presence, about heart murmurs or other functional manifestations is likely to arouse anxiety and fear which may result later in cardiac neurosis and excessive introspection. The child should not be labelled a cardiac invalid and allowed to remain one all his life, especially when his condition improves. Some parents, having been given the impression that the child has a fatal illness, are never able to accept the recommendations for increased or normal activity. Continuous conflict between the mother and child, endless anxiety on the mother's part, and lifelong invalidism are common in these cases.

PROBLEMS OF ADULTS

The seriously disintegrating influence on personal and family life and economic loss* to society due to heart disease in adults offer a challenge of the greatest magnitude. Some of the most distressing problems both for the patient and his family arise from prolonged financial or physical dependence on relatives or the community, inability to develop individual capacities or to realize ambitions for self or family, interruption in the continuity of normal pursuits and of usual human relationships, and the constant dread of impending physical disaster.

It has been estimated that over 80 per cent of chronic heart disease is due to syphilis, rheumatic fever, and the hypertensive and vascular diseases.[4] Of primary importance is the need for adequate continuous

*During the study of a group of cardiac patients, it was calculated that $25,235.79 was an approximation of the minimum cost to the community of care for 30 patients. This estimate included only actual hospital, nursing, and convalescent care. Public and private relief were factors in this cost when the patient's cardiac illness caused the family's dependency. Many items, such as outpatient care, medication, minor medical equipment of the sick room, etc., could not be accurately estimated, and were, therefore, excluded. The loss of earning power was not calculated, but was believed to constitute a very significant amount.

medical supervision for an indefinite period of time. The contribution of the social worker in making medical care possible and effective is well recognized. However, many communities still lack appropriate facilities for the care of convalescent patients and those with chronic heart disease.

Of equal importance with medical supervision is the need for regulation of the patient's daily activities. The adjustment of restrictions is influenced by many factors, viz., the personality of the patient, his relationships with the persons in his immediate environment, the conditions in which he works, the general economic situation, and the resources available in the community for the physically handicapped. After the physician and patient together have established the limits of the patient's physical activity, the patient should have a thoughtful interpretation of his condition which will place positive emphasis on his possibilities for attaining some of the satisfactions of life. The social worker and physician should collaborate with the patient in analyzing the patient's interests and needs so that the social worker may have the opportunity for significant constructive service to the patient and his family.

A recommendation for limited activity to a widowed housewife and conscientious mother of three small children, who must subsist on an income of $14 a week, has little likelihood of being carried out without knowledge of her specific duties, her attitude toward their execution, and her ability to delegate some of them to others. The problem of rehabilitation has received some attention in the literature on heart disease, but very little has been said about the problems of the housewife whose income is small. Her multiple activities often require effort more strenuous than that of many men in industry. Washing clothes, scrubbing floors, sweeping, bedmaking, carrying of bundles of food, hauling coal, cooking, etc., comprise a daily routine which cannot be postponed. Too often the patient has no relatives to share these labors or to protect her from the continuous emotional strains incident to the physical care of children, their susceptibility to disease, and the problems of their emotional and intellectual development. When resources within the family are lacking to help the homemaker or to provide a substitute for her, community agencies may furnish assistance or help to place children elsewhere temporarily. The traumatic effect on the emotional development of young, impressionable children of the continuous presence of a case of severe chronic heart disease in the home is not inconsiderable.[5]

The wage earner can occasionally be placed on a less strenuous job within the industry in which he is already employed. When this is not possible, re-education through the state department of rehabilitation is available under certain conditions; in such cases, age, previous

schooling, and adaptability are taken into consideration. The unwillingness of many industrialists to employ the physically handicapped often makes re-employment impossible.

A case[2] in point is that of a well-educated, 52-year-old Swiss hotel steward who had earned approximately $500 a month in fashionable hotels in Europe and America. His first cardiac illness occurred at the age of 47 years, and during the following two years his gradually increasing incapacity made it impossible for him to work and rendered him destitute. After he improved, following total thyroidectomy and social treatment, attempts were made to re-establish him in his former occupation. He was repeatedly refused work because employers feared that such a "poor risk" might increase their rate of workmen's compensation insurance. Although he was prepared to take a position as a "checker" in a restaurant, at a very small salary, he was rejected despite his willingness to sign a waiver of liability. For this patient work was found as a laboratory assistant, but for many others there is no such opportunity.

A fine alternative for many patients is available in communities which maintain workshops for the handicapped. The opportunity to work under sheltered conditions, without the usual industrial competition, to increase gradually the amount of work in proportion to gain in physical capacity, and to earn enough for self-maintenance has been a great boon to patients who might otherwise have become completely dependent and hopelessly invalided. The improvement in the condition of cardiac patients at work in the Project of the Handicapped carried on by the Federal Emergency Relief Administration in Boston justifies the hope that better economic conditions will make possible the establishment of more such projects, either under private or public auspices, with a wage rate which will insure financial independence.

In order to open up opportunities for the development of the patient's personality, appropriate recreation and other interests outside of work deserve more attention than they usually receive. Even if he has opportunities for work and recreation, the patient may not avail himself of them for fear of the adverse effects of the exertion involved. For the social worker, as well as for the physician directing the patient's treatment, an optimistic and encouraging attitude is essential. Experience has demonstrated that some patients, when relieved of undue anxiety, are capable of a life fuller and more effective than they had ever imagined possible.[6, 7] On the other hand, for the patients whose disease is not progressing, the temptation is great to cast aside the precautions for the avoidance of emotional strain and undue physical exertion. Many patients who have done well for years break down later because of unfavorable social influences. This "asymptomatic period"[8] offers the greatest opportunity for preventive work. Many physicians in private practice give this aspect of treatment considerable attention, but in busy clinics and hospitals it is the social worker's function to collaborate with the physician in assisting the patient to prevent or minimize disability. This requires understand-

ing of the patient and his illness, skillful and continuous treatment, and thorough knowledge of, and ingenuity in, the use of community resources.

An appreciation of the role that extreme emotional stress plays in the initial and recurrent attacks of both organic and functional heart disorders is necessary for adequate treatment. Alleviation or elimination of causes of emotional disturbances may increase the patient's activity and his ability to function as a member of society. A recent study[2] disclosed considerable evidence of the close interrelationship of the psychologic, social, and physical factors in patients' reactions to improved health. In some of these patients long continued illness, separation from normal adult activities, physical distress, reaction to dependency on relatives and community, and diminished resources due to the tremendous cost of chronic illness had produced fear and anxiety disabling to a degree out of proportion to the extent of the cardiac illness. Also, improved health in some cases did not reduce the patient's total disability. Within recent years, psychiatric and medical literature[9-15] has revealed an increasing interest in the study of the interrelationships of psychic and somatic factors in cardiac disorders. A greater awareness on the part of social workers of the dynamic aspects of these problems will enable them to recognize what social and emotional factors may be operating, and may help to show that suitable psychiatric and medical assistance is necessary for the reduction of disability and the enrichment of human life.

SUMMARY

Adequate care of patients with heart disease involves a recognition of the significance for etiology, recurrence, and chronicity, of emotional and social influences in the lives of the individual patients. The reaction of the patient to his illness may in certain cases be as important as the disease itself. Therefore, in addition to awareness of the lesion, attention should always be focused on the person with the lesion. A program of care is incomplete that does not give equal attention to the preventive aspects and the therapy. Prerequisites of such a program are regular medical attention for an indefinite period; avoidance of infection, emotional stress, and undue physical activity; adequate nutrition; and a plan of work and recreation suited to the individual's limitations and interests. Schooling and occupational therapy for children in institutions and in their own homes and sheltered workshops and recreational facilities for adults should be fostered by the community. Medical, social, and psychiatric services are required in many cases. These objectives are attainable when there exists a relationship of mutual understanding among physicians, patients, families, social workers, and all others who are trying to diminish the disability in persons ill with heart disease.

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A STUDY OF THE CHEST LEADS OF THE ELECTROCARDIOGRAM WITH AN EVALUATION OF THE POSITIONS OF THE PRECORDIAL ELECTRODE*

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SINCE the introduction of chest leads into clinical electrocardiography in 1932,[1] there has been an increasing number of studies relative to their use and interpretation.[2-12] It is only recently, however, that a standard classification of technique and nomenclature has been established. Specific recommendations have been made that for ordinary purposes Lead IV R or Lead IV F be employed in such a way that relative positivity of the apical electrode is represented in the finished curve by an upward deflection, and relative negativity of the apical electrode by a downward deflection, as is customary with the limb leads.[13]

Many data have been accumulated on the changes in the chest leads in heart disease, especially in cases of infarction of the myocardium and diffuse myocardial damage from coronary disease.[3, 5, 15] Much of the experience in the past has been gained by placing the right arm electrode near the apex and the indifferent electrode on the left leg. This lead is essentially an inverted mirror image of the newly recommended chest leads.[13] By inverting and reversing the old tracing and looking at it in front of a strong light the image will show through the paper and will resemble that of the new chest leads.

Several authors have shown that a shift in the position of the heart may affect the axis deviation.[16, 17, 18, 23, 24] The direction of the extrasystoles caused by stimulating each ventricle may be changed by shifting the chest electrode from left anterior to right anterior thorax or from the left to the right of the spine, posteriorly.[17a] The effect on the electrical axis of rotation of the heart on its longitudinal axis has been demonstrated by Meek and Wilson.[22] In 1930 Wilson[19] showed that it is the electrode near the heart which determines the form of the ventricular electrocardiogram and that it matters little where the second electrode is placed, providing it is sufficiently distant from the heart.[4] Johnston, Kossmann and Wilson[2] also stated that when the distant electrode was placed on the hind leg of a dog, there was, in most of the experiments, a striking resemblance between the precordial curves and curves taken with the exploring electrode placed directly on the heart. More con-

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sistent similarity was found when the indifferent electrode was a central terminal connected through equal resistances to the forelegs and left hind leg. Wood, Bellet, McMillan and Wolferth[3] have pointed out that variations in the character of the tracing can occur if the precordial electrode is allowed to be displaced.

The authors, in a previous study of the significance of absence or small size of the initial positive deflection in the precordial lead,[14] noted that there were often discrepancies in the records of the same patients taken at different times. This was especially frequent in patients with large hearts, and the difficulty seemed probably related to the position of the precordial electrode on the chest. Accordingly, one of us (J. B. V.) thought that it would be of value to check a number of patients with enlarged hearts, using several positions, both inside and outside the apex, for the precordial electrode. Lead IV R was first chosen because we had been using this lead routinely for some months prior to the time when its official adoption was recommended.

In an endeavor to elucidate the variations mentioned above and to evaluate the new precordial leads (IV R, IV F, IV L) recently recommended by the committees of the American and British Heart Associations,[13] multiple precordial leads were made in a series of 67 patients.

METHODS

One of us (J. C. E.) personally studied all of the patients and placed the electrode on the chest after marking and measuring the points of placement. Records were made with a circular electrode, 3 cm. in diameter, from six different positions on the anterior chest wall, employing Leads IV R, IV F, IV L. (Fig. 1).

These positions are designated[13] CR, CF, and CL, according to the lead used (IV R, IV F, or IV L). To illustrate: Lead IV R is taken with the precordial electrode at the right margin of the sternum (CR_1), at the left margin of the sternum (CR_2), midway between the left margin of the sternum and the left midclavicular line (CR_3), at the left midclavicular line (CR_4), at the left anterior axillary line (CR_5), and at the left midaxillary line (CR_6). The subscripts mean that for the sternal leads the precordial electrode has been placed in the fourth intercostal space and that for the other leads it has been placed upon a line drawn from the left sternal margin in the fourth intercostal space to the outer border of the apex beat (or to a point at the junction of the midclavicular line and the fifth intercostal space) and continued around the left side of the chest at the level of the apex beat or of the junction mentioned. (Fig. 1.)

However, in the case of Lead IV R or IV F it has been recommended[13] that the precordial electrode be placed at the outer border of the cardiac apex regardless of the position of the apex with reference to the bony landmarks of the chest. For CF_4 or CR_4, this electrode is placed in the midclavicular line even when the cardiac apex is far to the left of this position. When there is no cardiac enlargement, position CR_4 or CF_4 may be the same as Lead IV R and IV F. In some cases in which there is cardiac enlargement, however, Leads IV R and IV F are the equivalent of CR_5 and CR_6, or CF_5 and CF_6. In taking Lead CR, the left arm wire was connected to the precordial electrode, the right arm wire to the right arm electrode, and the lead switch placed on Lead I, as in taking Lead IV R. In taking Lead CF, the left leg wire was connected to the precordial electrode, the left arm wire to the left leg electrode, and the lead switch was placed on

Lead III, as in Lead IV F. In taking CL, the left leg wire was connected to the precordial electrode, the left arm wire to the left arm electrode, and the lead switch was placed on Lead III as in Lead IV L.*

When the patient under observation was confined to bed, the head of the bed was raised to an angle of 60° to correspond to the angle of the chair used in the heart station for the ambulatory patients. To avoid overshooting, the resistance of the skin was lowered to 2,000 ohms, or less, by careful preparation. In all records the standardization was 1 mv. = 1 cm. In all of the cases studied, the position of the apex was ascertained by the usual clinical methods. If its position could not be located by these means, it was determined by the roentgenogram. Nearly every patient had an orthodiagram or teleoroentgenogram, or both, in order to check the clinical observations. In most instances several electrocardiograms were taken at different times, using multiple precordial leads in each instance. These tracings checked closely with one another except in cases of recent coronary occlusion in which there were the usual changes seen in such serial records.

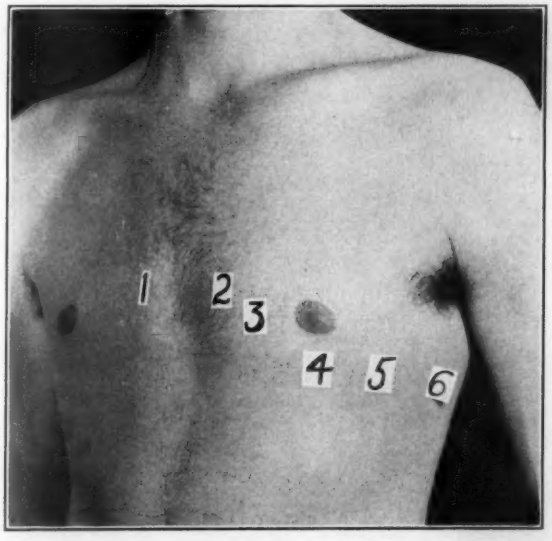

Fig. 1.

Since it had been previously determined[7] that the configuration of the chest in normal boys has no particular correlation to the T-wave pattern in the chest electrocardiogram, as it does in the limb leads, we did not make thoracic measurements in this study.

ANALYSIS OF DATA

The etiological diagnoses in this group of 67 cases are given in Table I. There were 10 subjects with hearts of normal size and function and with no axis deviation (9 adults and 1 child of 13 years). Four patients had no appreciable cardiac enlargement, although some axis deviation was present (left in two cases and right in two cases). Cardiac enlarge-

*It is to be noted that the directions given by the Committees of the American and British Heart Associations for taking Lead IV R say to connect the left leg wire to the precordial electrode, the right arm wire to the right arm electrode, and to place the lead switch on Lead II. Our method of placing the left arm electrode on the precordium and leaving the right arm electrode in place, using Lead I switch, yields exactly the same results and is slightly more convenient, in our opinion.

ment was present in the remaining 53 subjects. Twenty-five patients whose apices lay at the anterior axillary line were studied. Four of these had had myocardial infarction. There were 7 patients who had right axis deviation in the electrocardiogram and moderate cardiac enlargement. Ten patients with coronary thrombosis had moderate cardiac enlargement. The remaining 11 patients had slight or moderate cardiac enlargement as the result of syphilitic aortic regurgitation, hypertension and arteriosclerosis, or chronic rheumatic heart disease.

TABLE I

ETIOLOGICAL DIAGNOSIS

	NUMBER OF CASES
Myocardial infarction	16
Rheumatic heart disease	17
Arteriosclerotic heart disease (7 with hypertension)	16
Congenital heart disease (2 under 12 years of age)	4
Syphilitic heart disease (aortitis)	4
Normal adults (1 child 13 years old)	10
Total	67
The Same Cases Arranged According to the Size of the Heart	
Normal	10
Slight cardiac enlargement (2 with congenital heart disease) (2 with myocardial infarction)	4
Apex at anterior axillary line (4 with myocardial infarction) (6 with right axis deviation)	25
Right axis deviation (Moderate CE*)	7
Myocardial infarction (Moderate CE*)	10
Miscellaneous group (Slight to moderate CE*)	11
Total	67

*Cardiac enlargement.

Normal Subjects.—Each of the 10 normal subjects had a diphasic QRS complex in all positions of the precordial electrode, although in midaxilla the R-wave measured 15 to 25 mm. and the S-wave 0 to 5 mm. The R-wave (initial positive deflection) gradually increased in positivity (height) from the sternum to the left axilla. In none of these cases was there any sudden shift in direction of the QRS complex (Fig. 2). The initial positive deflection (R) varied from 2 to 20 mm. at CR_2 (left border of sternum) and from 2 to 10 mm. at CR_1 (see Table IIA). This agrees with the results of Shipley and Hallaran's study[8] of 21 normal subjects.

Patients With No Cardiac Enlargement.—In 2 patients with old anterior infarction and left axis deviation (but no appreciable cardiac en-

largement), the R-wave was absent at positions C_1, C_2, and C_3 in all three chest leads (CR, CF, and CL). However, in one of these cases R was 22 mm. at C_4, 20 mm. at C_5, and 16 mm. at C_6; in the other, it was absent at C_4, 10 mm. at C_5, and 7 mm. at C_6 (Table III B). Two children without appreciable cardiac enlargement had right axis deviation. One of these had rheumatic heart disease with mitral stenosis, and the other had congenital pulmonic stenosis. In the former, R increased gradually and S decreased as the electrode was moved toward the axilla; in midaxilla the S almost disappeared. In the other case the S was completely lost at

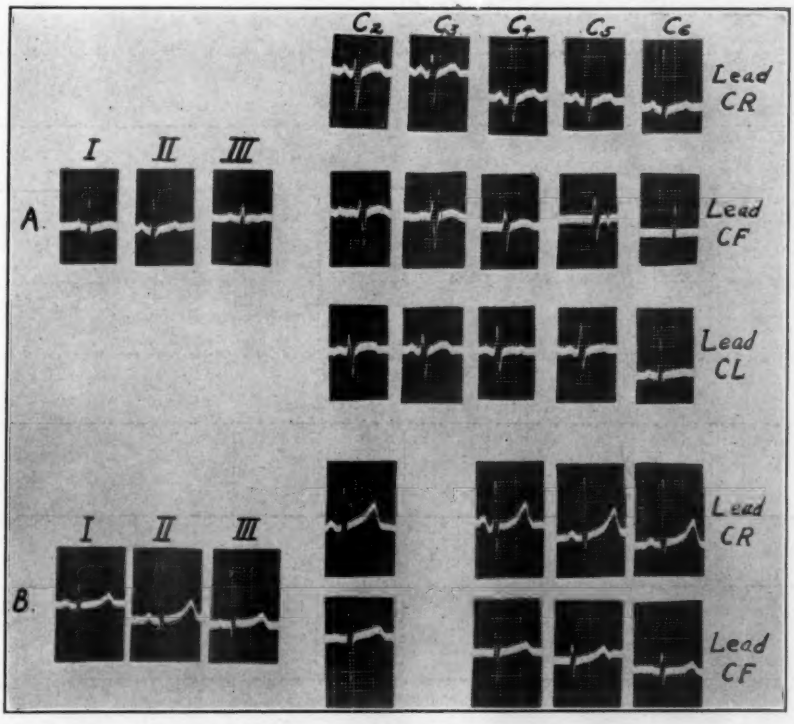

Fig. 2.—Multiple precordial leads of patients with normal hearts.

A, A comparison of Leads CR, CF, and CL in various precordial positions. Position C_1, right edge of sternum 4th intercostal space (not shown because of similarity to C_2). C_2, left edge of sternum 4th intercostal space. C_3, midway between margin of sternum and left midclavicular line. C_4, left midclavicular line at 5th intercostal space. C_5, anterior axillary line. C_6, midaxillary line. Note the greater amplitude of all waves in Lead CR and the gradual change in the QRS complexes with increase in R-waves and decrease in S-waves as the electrode is moved laterally.

B, a comparison of Leads CR and CF, [CL (not shown) nearly identical to CR]. Note the more satisfactory P- and T-waves in Lead CR.

position C_6 (midaxilla). The R-wave was absent in positions 1, 2 and 3 in this case, but appeared in position 4 and was large at C_5 and C_6.

Patients With Cardiac Enlargement.—(1) Apex at anterior axillary line: In 21 cases (excluding 4 cases of myocardial infarction), the apex was located at the anterior axillary line (Table III C). In 14 of these a sudden shift in the direction of the main deflection of the QRS oc-

curred at the midaxillary position (Fig. 3*A*). In nine of these 14 cases there was some gradual decrease in amplitude of the S-waves as the midaxillary position was approached. At this point there was then a sudden shift to a large R-wave without any S-wave. In one case the S-wave and the R-wave gradually increased until, at C_6, there was a

TABLE II

AVERAGE OF QRS AND T DEFLECTIONS IN MILLIMETERS

POSITION	C_1	C_2	C_3	C_4	C_5	C_6	AXIS DEV.
				A			
				10 Normal Subjects			
R	6	10	14	17	18	18	All
S	7	9	9	7	5	2.5	with
T*	+2	+5	+6	+5	+4	+3	none
				B			
				4 Patients With Abnormal Hearts But Without Cardiac Enlargement			
R	R_0Q_6	R_0Q_7	Q_3R_4	11	15	11	Two with old anterior infarctions — Left axis deviation
S	0	0	0	2	1	1	
T	1+	1+	1+	1+	1+	1+	
	1–	1–	1–	1–	1–	1–	
R	15	15	16	21	30	19	Two with pulmonary stenosis — Right axis deviation
S	5	14	10	9	0	0	
T‡	Both +	+	+	+	+	+	
				C			
				21 Patients With Apex at Anterior Axillary Line†			
R	1.5	3	3	6	12	14	11 Left axis deviation
S	7	14	15	15	11	0	6 Right axis deviation
T‡	12+	13+	15+	12+	11+	5+	4 No axis deviation
	9–	8–	6–	9–	10–	16–	
				D			
				5 Patients With Acute Anterior Infarction			
R	R_0Q_{21}	R_0Q_9	$0\ Q_{11}$	$0\ Q_{12}$	$0\ Q_8$	$R_{10}Q_{-6}$	4 Left axis deviation
S	0	0	0	0	0	1	1 No axis deviation
T	4–‡	1+	1+	2+	2+	5–	
	1+	4–	4–	3–	3–		
				E			
				7 Patients With Old Anterior Infarction			
R	$0\ Q_4$	$0\ Q_8$	$0\ Q_6$	$R_0Q_{0\text{-}6}$	$R_0Q_{1\text{-}4}$	R_8Q_0	5 Left axis deviation
S	3	4	4	6	0	3	2 No axis deviation
T	5+‡	5+	5+	5+	5+	1+	
	2–	2–	2–	2–	2–	6–	

*All T-waves upright in this group; average height given.
†Four with myocardial infarction not included.
‡Number of upright (+) and flat or inverted (–) T-waves.

sudden change to a larger R and absent S. In all of these records the R-wave was over 10 mm. when the S-wave was absent or low in position C_6. In 6 of the 21 cases in which the apex was at the anterior axillary line (in 2 of which right axis deviation was present), there was a gradual decrease in the amplitude of the S-wave until position C_6 was reached, when the S became very small (1 to 3 mm. in height, with an R of 9 to 23 mm.).

In five of these 21 cases the major change in the R/S ratio occurred at the anterior axillary line, with the R and S in the axilla (C_6) of about the same amplitude as at C_5 (Fig. 4*A*). The S was absent at C_5 in 2 cases, and in 3 there was a small S (2 to 8 mm.) with a large R, the S disappearing at C_6. In only 2 of these 21 cases was there no marked shift in QRS, and in one of these left bundle branch block was present.

In 6 of the 21 cases the R-wave was small, from 1 mm. to 5 mm. in the first five positions, becoming larger in midaxilla. A large S-wave of 10 mm., or over, was present in the first five positions of the precordial electrode. In all of these there was clinical evidence of myocardial damage of severe degree. Only one patient with the apex at the anterior

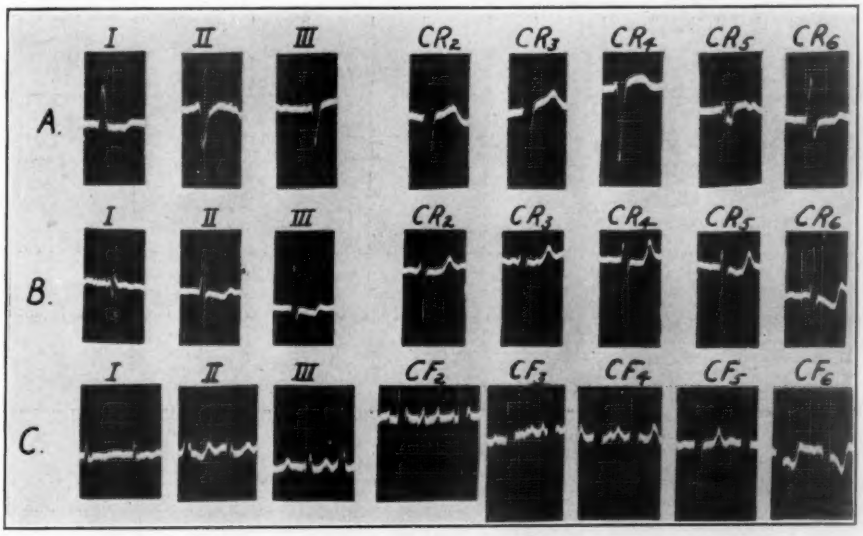

Fig. 3.—Variations in the chest lead with shift of the precordial electrode in patients with the cardiac apex at the anterior axillary line.

A, Case A.P. Hypertensive and arteriosclerotic heart disease. Note the marked change in the QRS complex at positions 5 and 6.

B, Case M.B. Rheumatic heart disease. Observe the striking shift of the ventricular complex at position 6.

C, Case J.L. Rheumatic heart disease; auricular flutter with varying degrees of A-V block. A similar but less striking shift is seen at C_6. This case also illustrates the value of medial precordial leads in demonstrating the auricular waves when these waves are shown poorly in the limb leads. Lead CR (not shown) was practically identical with lead CF.

axillary line had no R-wave in the first 5 positions. He had an old anterior myocardial infarct, but his precordial electrocardiogram showed an R-wave at C_6.

(2) Apex 2 cm. beyond the midclavicular line: In two cases of cardiac enlargement and left axis deviation there was no great shift, but in one there was a Q-wave of 1 mm. and a diphasic R-S at positions C_1 and C_2 in CR, CF and CL. The T-waves were negative in C_1 and C_2 but upright in C_3, C_4, C_5, and C_6. In one other case in which there

was a tendency to right axis deviation, R increased at the apex, but no S-wave appeared in any of the precordial leads.

Patients With Right Axis Deviation.—There were 13 cases of right axis deviation, and in 7 of these there was a sudden change to a large R and absent S as the electrode was moved from the anterior axillary line to the midaxillary position (six of these seven were cases in which the apex lay at the anterior axillary line and were also included in the study of that group) (Fig. 2*B* and Table III).

TABLE III

AVERAGE OF QRS AND T DEFLECTIONS IN MILLIMETERS

	CR_1	CR_2	CR_3	CR_4	CR_5	CR_6
			A			
			13 Patients With Right Axis Deviation			
R	4	6	7	8	8	13
S	6	14	15	7	8	3
T*	9+	6+	9+	7+	7+	6+
	4–	7–	4–	6–	6–	7–
			B			
			6 Patients With Right Axis Deviation and Apex at Anterior Axillary Line			
R	3	5	5	8	6	12
S	4	11	18	10	14	3
T	–*	1+	3+	3+	3+	1+
	3–	5–	3–	3–	3–	3–
			C			
			7 Patients With Right Axis Deviation and Only Moderate Cardiac Enlargement			
R	5	8	11	15	10	12
S	2	13	11	9	0.6	1
T	3+*	4+	4+	4+	4+	3+
	1–	2–	2–	2–	2–	3–

TABLE IV

J. H., SITUS INVERSUS WITH DEXTROCARDIA

	CR_1	CR_2	C_3	C_4	C_5	C_6
R†	6	3	2	0.5	0.5	0
S	24	23	25	18	15	11
T	+	+	diphasic	diphasic	diphasic	diphasic
R‡	9	7	15	20	23	27
S	27	28	23	13	11	0
T	+	+	+	diphasic	diphasic	diphasic

*Number of upright (+) and flat or inverted (–) T-waves.

†Precordial electrode on left anterior thorax.

‡Precordial electrode on right anterior thorax, with C_1 left sternum, C_2 at right sternum, etc.

In patients with right axis deviation but without appreciable cardiac enlargement, the greatest change occurred at position C_5, where the R became larger and the S smaller. This was much like the changes found in those having hearts of similar size without right axis deviation. In three cases there was a gradual shift as the electrode was moved laterally, and in 3 others there was little change in the character of the QRS in the various positions. One of these last cases was that of a boy of 19

years with the tetralogy of Fallot and marked right axis deviation. One patient with situs inversus and dextrocardia was studied with multiple precordial leads on both the right and left sides of the chest. In this patient the height of the R increased as the precordial electrode was placed in the six corresponding positions on the right chest, but the opposite occurred when the records were made with the electrode in the usual six positions on the left side of the chest. The P-waves were inverted in CF and CR when the chest electrode was on the left anterior thorax but were upright in CL. When the multiple precordial leads were taken on the right side, the P-waves were upright in CF and CR but inverted in CL (Table IV).

Patients With Coronary Thrombosis.—There were 16 patients with old or recent myocardial infarctions on whom multiple precordial

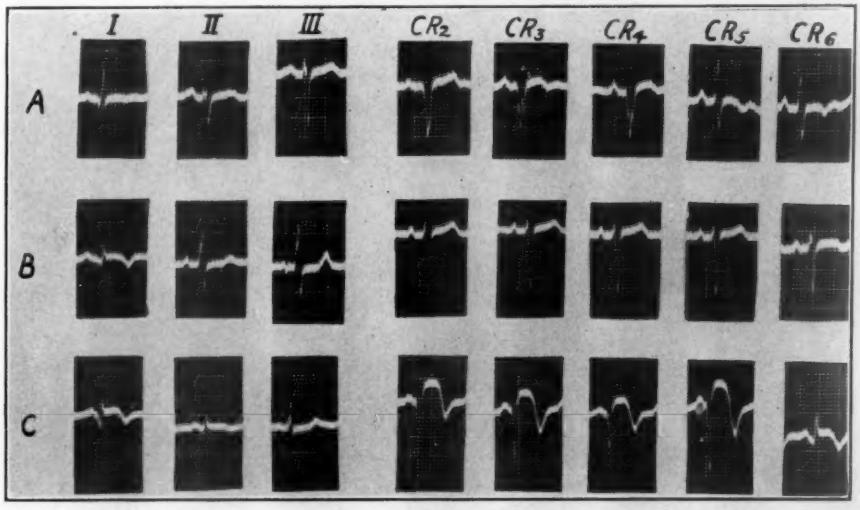

Fig. 4.—Variations in the precordial electrocardiograms of patients with anterior myocardial infarction.

A, Case C.S. Old anterior infarction with marked cardiac enlargement. Note the marked shift in the QRS and T-waves at positions 5 and 6.

B, Case M.F. Old anterior infarction with little cardiac enlargement. This was one of the 2 patients in whom there was no essential change in QRS complex in the various positions.

C, Case E.D. Recent anterior infarction with moderate cardiac enlargement. There is a striking change in the QRS complex at position 6, with loss of the deep Q-wave.

leads were made. Only 2 of these patients had posterior infarctions alone. On 7 who had had recent anterior infarcts serial studies were made; 7 had old anterior infarcts. In 4 of the 14 cases with anterior infarction the cardiac apex was at the anterior axillary line. In these 4 the R-waves were absent in the precordial lead in the positions medial to the apex (Fig. 4*A*). In only one case, however, was the R-wave absent in the midaxillary position (C_6). Five patients had moderate cardiac enlargement, and two of these showed relatively little change in the QRS in any of the six positions. Seven of the 16 patients with

coronary thrombosis had slight or no cardiac enlargement. None of these patients had an R-wave in the first three positions. One began to have an R-wave at the apex, and three lost the initial Q-wave and had the initial positive deflection (R) at midaxilla. Two of these 7 developed the R at the anterior axillary line, having had no R at C_1, C_2, C_3, or C_4. Only one patient had a Q-wave (no initial R) in all positions of the precordial electrode. He had had three previous myocardial infarcts, one anterior and two posterior.

In 8 of the entire 67 cases, the R-wave was absent or small at CR_4, CF_4, and CL_4. It was absent in 5 cases of anterior myocardial infarction and small (2 mm.) in all positions in a case of old myocardial infarction of the anterior type (Fig. 4*B*). The R was small (2 mm.) in one case of myocardial damage in which there was a recent posterior infarct. In one case of recent anterior infarction the R was absent in IV R, IV F, and IV L in all positions of the precordial electrode except C_6, where a 6 mm. R with no S occurred (Fig. 4*C*). The apex was just outside the midclavicular line in this case. In a similar case there was a Q of 2 mm. and an R of 7 mm. in midaxilla, with a larger Q and no R in all other positions. In both of these latter cases the T remained negative (abnormal), and the RST segments were elevated with all positions of the precordial electrode except the midaxillary.

Miscellaneous Group.—In 4 of 11 cases not classified in the previous groups, the apex was from 1 to 2 cm. beyond the midclavicular line. Most of the changes in amplitude of R and S occurred at the midclavicular line (C_4). In the remaining 7 patients, who had myocardial damage, there was a gradual loss of the S-wave with increasing amplitude of the R-wave as the precordial electrode was moved laterally.

A COMPARISON OF LEADS CR, CF, AND CL

In studying the relative merits of the various chest leads, the data from all 67 cases were available. In 7 cases leads CR and CF only were taken, and were essentially the same. In 60 of the patients (10 with normal and 50 with abnormal hearts), leads CR, CF, and CL were taken from the various precordial positions. In 15 of these patients there was no significant difference in the three leads. In 22 cases, however, it was felt that CR was more satisfactory than CF or CL (i.e., it most closely interpreted the clinical picture). In 8 patients CR and CF were similar, and better than CL, the main difference being absent R-waves in several positions, less amplitude of QRS and inverted P-waves in CL.

In only 3 of the 50 patients with abnormal hearts, on whom CR, CF and CL were taken, was CF considered more satisfactory than CR and CL (because of greater amplitude of QRS and T).

In the 12 remaining cases, CR (likewise Lead IV R) seemed more desirable because of the relatively higher amplitude and fewer small R-waves at the midclavicular line in the fifth interspace.

In no instance was CL considered superior to CR or CF.

All of the leads were similar in 4 of the 10 subjects with normal hearts who were used as controls. In 2 the only difference was a greater amplitude of all waves in CR than in CF and CL (Fig. 2). In the other 4 there were flat and diphasic T-waves and inverted P-waves in several positions of Leads CF and CL, but CR was normal.

In 4 cases the QRS did not shift in the midaxillary position with CL, but did with CR and CF. In one case it shifted with CR but not with CF; Lead CL had a diphasic QRS in that position.

In 3 patients, none of whom had myocardial infarction, the R was 2 mm. high in CR_4 but absent in CL_4. With CF the R was absent at C_1 and C_2 in one of these cases, and present at the other positions, where it was similar to CR but of less amplitude.

The Q-waves were deeper in CF and CL than in CR in one patient with anterior and posterior myocardial infarcts. One patient with congenital pulmonary stenosis and little myocardial damage had a 3 mm. Q-wave in CL_6. Another had no shift of QRS in CF_6, but did show it in CR and CL.

In general, Lead CR (likewise Lead IV R) seemed more desirable because it yielded higher amplitude of the QRS complexes. With but few exceptions, the Q-, R-, and S-waves tended to be of greater amplitude in Lead CR than in CF or CL. This factor may be of importance in interpreting the electrocardiograms both of normal persons and those with damaged hearts. For example, a small initial positive deflection (R-wave) is usually regarded as an abnormal finding,[14, 26] but in some of our cases the R-wave was within normal limits (over 2 mm.) in Lead CR and was abnormal (less than 2 mm.) in Lead CF or CL, or both, when taken at the same point on the precordium. Obviously, both results could not be correct, and in most instances of this type Lead CR seemed to be more accurate when correlated with the clinical findings.

There were three deaths among the patients in this series, and a postmortem examination was done in each case. In Case F. D. there was an old infarct at the apex; the heart weighed 635 gm. Clinically, the apex impulse had been in the sixth left intercostal space 3 cm. beyond the midclavicular line. The limb leads showed slight left axis deviation. The precordial leads exhibited a shift from a deep Q-wave and no R-wave in all 5 medial positions to a large R-wave at position C_6 (25 mm. at CR_6, 15 mm. at CL_6, and 14 mm. at CF_6). This type of change is illustrated in Fig. 4*C*.

This sudden change at position C_6 from an abnormal complex to a normal one was frequently seen in the patients with anterior myocardial infarction. In Case L. M. there was an old thrombosis of the right coronary artery with diffuse scarring of the posterior surface of the left

ventricle. The heart weighed 510 gm. There was right axis deviation with deeply inverted T-waves in Leads II and III. The cardiac apex had been located 2 cm. beyond the midclavicular line in the sixth left intercostal space. The precordial leads had a normal appearance except for negative T-waves at CR_3, CR_5 and CR_6. The shift in the QRS complex occurred at C_5, where the S-wave disappeared, and the R-wave increased in height. This shift at C_5 was commonly found in patients with moderately enlarged hearts and did not represent a significant change in many cases.

In Case C. Sl. the patient had rheumatic heart disease with mitral stenosis. The heart weighed 410 gm. Clinically, the cardiac impulse was located in the sixth left intercostal space 2 cm. beyond the midclavicular line. Roentgenograms revealed cardiac enlargement to the right and left with a large pulmonary conus. The electrocardiograms showed right axis deviation with flat T-waves in the limb leads. The precordial leads had an R- and S-wave in all positions except that S was absent at C_1. The T-waves were all inverted. There was the usual gradual shift of the QRS until, at C_6, the major portion of the QRS complex was above the isoelectric level.

In general, the T-waves were of greater amplitude in Lead CR than in Lead CF or CL. They were usually largest with the electrode at position C_4 in both normal subjects and patients with heart disease. The P-waves were largest and most distinct when the precordial electrode was at position C_2. In some cases the P-waves were more distinct in the precordial lead at position C_2 than in the limb leads. In a few cases, the P-waves were inverted in CF (and IV F) when they were normal in CR and CL (also IV R and IV L).

DISCUSSION

The Normal Lead IV.—In the standard Lead IV[13] the P-wave is upright, and with the precordial electrode at the apex it may be smaller than in the limb leads. If the precordial electrode is moved medially to the left edge of the sternum (fourth intercostal space), the P-wave becomes larger and may exceed that in the limb leads. In certain cases, as in auricular flutter, a lead at this point may be of value in bringing out the auricular waves. However, in this respect only did we find the sternal lead more satisfactory or superior to the precordial leads with the electrode at the apex.

The QRS complex is usually of greater amplitude than in the limb leads, frequently being diphasic, with an R- and S-wave. The R-wave often predominates and may normally be the only wave present. With great amplitude of the R-wave a small Q-wave (1-2 mm.) is seen occasionally and is not of pathologic significance. An R-wave of 2 mm. or less is usually abnormal if the subsequent S-wave is 10 mm. or more

in depth. The significance of absence or small size of the initial positive deflection (R-wave) in the precordial lead has been emphasized by a number of writers, including ourselves.[5, 14, 26]

The T-wave is positive and as a rule sharply peaked, sometimes reaching a height of 10 mm. Very high T-waves in this lead are considered abnormal by some authors, regardless of their direction.[25] The isoelectric R-T segment is of short duration and may occasionally be elevated or depressed slightly (1-2 mm.). Digitalis depresses the R-T segment, as in the limb leads, but frequently to a much greater degree.

As determined by our studies with Leads IV R, IV F, and IV L, there is no characteristic change in the QRS complex associated with either right or left axis deviation or ventricular preponderance, which confirms Willcox and Lovibond's observations[6] with an apex-right arm lead (now known as Lead IV R).

Multiple Precordial Leads.—Our findings agree with those of Sorsky and Wood[7] in so far as the two studies are comparable. Using three positions for the precordial electrode, designated as apex, right, left pectoral (old Lead V—precordial electrode and left leg), they found that within certain limits shift of the exploring electrode to the right of the apex yields a relatively smaller initial positive deflection and a less inverted T-wave. Shift of the exploring electrode to the left tends to have the opposite effect. They found a small Q in the apex-right arm lead in 30 of 150 normal subjects, and its counterpart, a small R, with the old apex-left leg lead, in 20 of 150 normal subjects. On the whole, we agree with the conclusions drawn by Shipley and Hallaran[8] from their study (using three positions of the precordial electrode) of 21 normal persons, that alterations of the simple QRS form are much more frequent in lateral than in medial positions. The more distant the chest electrode is from the heart laterally, regardless of size or axis deviation, the smaller the S becomes and the greater the R/S ratio. The total amplitude of the QRS becomes smaller as the precordial electrode is moved from the apex, whether in a medial or lateral direction. The height of the R-wave increased as the precordial electrode was moved laterally from the sternum in all of our cases except a few with anterior myocardial infarction. Even in some of these an R-wave appeared at C_5 and C_6 (replacing an initial Q-wave), and in two an R appeared at C_4. With left bundle branch block the R-wave may be small or absent, and the RS-T segment elevated in all positions except C_6. There was an abrupt shift in the R/S ratio (large R and small or absent S) at C_5 in most patients with moderately enlarged hearts, and at C_6 in those whose hearts extended to the left anterior axillary line or beyond. All of the normal subjects had some change in the QRS complex in the multiple positions. There were four of the 57 patients with abnormal hearts whose QRS complexes did not change appreciably when the precordial electrode was moved from one position to another. Only

five patients had no initial positive deflection of the QRS at all of the positions, and these were all cases of anterior infarction. Two patients had R-waves of approximately 2 mm. in all positions.

In persons with normal hearts we found, as did Shipley and Hallaran,[8] that the T-wave tends to be largest with the precordial electrode at the apex. In our series the most satisfactory position for recording the T-waves was C_4 (midclavicular line), with the right arm as the indifferent electrode (CR). Hall[21] found this position (Lead IV R) reliable in studying cases of anterior myocardial infarction and noted that the T-waves in this lead were often still negative after the T-waves in Lead I had returned to normal.

The P-waves were largest and most distinct at position C_2 and more satisfactory with Leads CR and CL than CF. Because of variations in the P-waves and a tendency to small initial positive deflections of the QRS at the sternal position, we do not feel that this position is desirable for routine use, although it might occasionally be of value for special studies in conditions such as auricular flutter (Fig. 3*C*).

One case not included in this series was of interest in connection with the various precordial leads. A patient with purulent pneumococcic pericarditis (diagnosis substantiated at autopsy), showed the electrocardiographic pattern typical of acute pericarditis (elevation of the RS-T segments in all of the limb leads). Lead IV R showed a striking elevation of the RS-T segment which persisted on serial examinations. Vander Veer and Norris[27] found no significant abnormalities in Lead V (Wolferth and Wood, apex and left leg) in 4 cases of purulent pericarditis in which there were striking changes in the limb leads. Bellet and McMillan,[28] who studied patients with pericarditis by means of Leads IV, V, and VI (old terminology), found RS-T deviations in all of these leads in some cases, but it was most striking in Leads IV and VI. The logical explanation of this finding (i.e., little or no change in Lead V in pericarditis) is that the algebraic summation of the deviations of the RS-T segments in Leads IV and VI is approximately equal to that of Lead V. Thus, with depression of the RS-T segment in old Lead IV and elevation of the same segment in Lead VI, Lead V would tend to be isoelectric in these cases. For this reason, Lead IV F (the inverted mirror image of old Lead V, and taken at identical points) is inferior to Lead IV R as a diagnostic aid in this condition (Fig. 5).

There are probably several factors which affect the size and contour of the QRS complex in the precordial leads. Analysis of our data suggests that the size of the heart (enlargement) and the position of the precordial electrode are two very important ones. With normal hearts there were only gradual and slight changes in the QRS complex as the precordial electrode was moved laterally. The most striking changes occurred in the patients with enlarged hearts when the precordial electrode was placed on or outside the apex. In several of these, definitely

abnormal precordial electrocardiograms in the medial positions (which interpretations concurred with the clinical picture) became normal when the electrode was placed at or just outside the apex (Fig. 4*A*). This shift of the QRS complex occurred with both right and left axis deviation when the heart was enlarged.

Our studies suggest that ordinarily it is desirable to place the precordial electrode at the *position of the apex* (midclavicular line, fifth left intercostal space) in patients with *normal* or only *slightly enlarged hearts.* However, in patients with *moderately* or *greatly enlarged hearts,* we feel that the electrocardiogram will more often substantiate the clinical diagnosis if the precordial electrode is placed *medial to the apex* (left border of dullness), *and never farther to the left than the anterior*

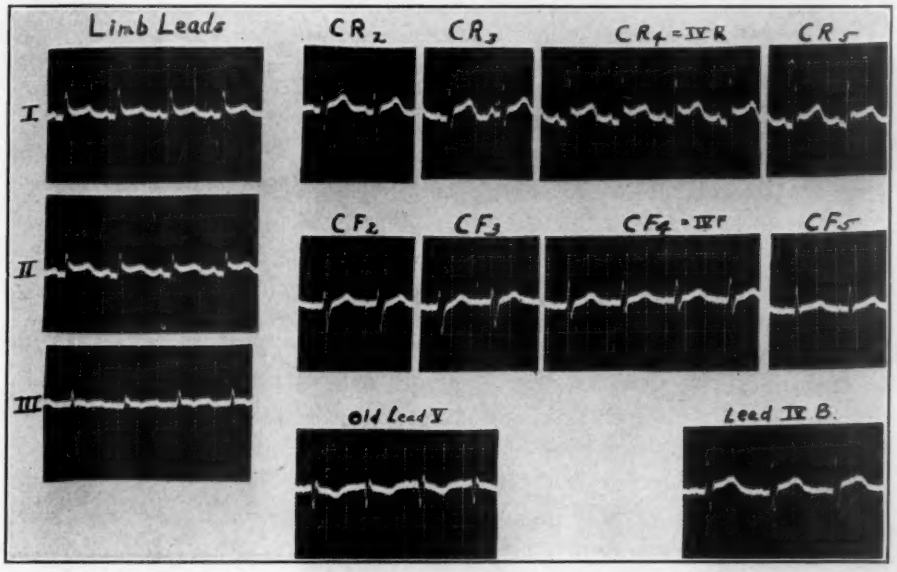

Fig. 5.—Case M.L. White woman 67 years of age. Pneumonia followed by pericarditis. Pericardial tap (350 c.c.) revealed a seropurulent fluid with a few gram-positive cocci and *Staphylococcus aureus* on culture. This record, taken during the acute stage, shows the typical pattern of acute pericarditis in the limb leads. In the precordial leads it is quite evident that Lead CR is superior to Lead CF in demonstrating the acute changes (elevation of the R-T segments). This change is most striking when the electrode is placed at the apex area (CR_4 or IV R). Practically no R-T segment change is present in Lead CF at any precordial position. Lead CL in the same positions showed no R-T deviations. Old Lead V and Lead IV B likewise show no change of diagnostic significance.

axillary line. This point of view is not in keeping with the recent recommendations[13] for precordial leads, but we believe that the results will be more satisfactory in cases in which the heart is enlarged if this rule is followed. The whole question of the optimum position of the precordial electrode for routine use is not settled; it is very probable that for best results the cases will have to be individualized and perhaps more than one position utilized in many instances. Certainly, in the study of some pathologic conditions, such as pericarditis and infarc-

tion of the lateral wall of the left ventricle, no one lead can give completely satisfactory results, and in cases of this type exploration of several precordial areas is often indicated.

Roth[4] points out that in 1900 Einthoven and de Lint[29] were searching for a lead that would yield the largest possible deflections in the electrocardiogram and came to the conclusion that this requirement was fulfilled best by the right-arm-apex lead now known as Lead IV R. This lead yields maximal auricular and ventricular complexes with the galvanometer string at standard sensitivity. In the majority of instances, in our experience, Lead CR_4 fulfills these conditions better than CF_4 or CL_4. It seems to us that if a single precordial lead is to be taken routinely, Lead CR_4 (midclavicular line, fifth intercostal space, and right arm) is the most desirable.

SUMMARY

1. Multiple precordial leads were studied in 67 persons. Ten of these had normal hearts and the remainder were suffering with cardiac disease of various kinds. The recent recommendations of the American and the British Heart Associations for routine and multiple precordial leads were followed. Leads IV R, IV F and IV L were taken with the precordial electrode just outside the apex and the indifferent electrode on the right arm, left leg, and left arm, respectively. The galvanometer connections were made in such a way that relative positivity of the electrode nearer the heart was represented by an upward deflection (R-wave). The multiple leads, designated CR, CF, and CL, were taken with the precordial electrode in six different positions extending from the right sternal border to the left midaxillary line (C_1, C_2, C_3, etc.). The normal Lead IV and its variations are discussed.

2. In medial positions (near the sternum) the initial positive deflection (R-wave) tends to be small, and the following S-wave large. As the precordial electrode is moved laterally, the size of the R-wave tends to increase and that of the S-wave to decrease. In normal hearts this change in the QRS complexes is slight, but, when the heart is enlarged, the shift of the QRS is often striking, and may be quite sudden and marked with only a slight change in position of the precordial electrode. A complete change of the QRS complexes from abnormal in the medial positions to normal in the axillary positions was seen in several of the patients with diseased, enlarged hearts. This was especially evident in those who had previously had anterior myocardial infarcts. There was no relation of this shift to the electrical axis in the limb leads.

3. The T-waves were usually most satisfactorily recorded with the exploring electrode near the usual apex area (5th intercostal space, left midclavicular line). The P-waves, in most instances, were largest when the electrode was placed in the medial positions, just to the left of the sternum.

4. All waves tended to be of greater amplitude with Lead CR than with Lead CF or CL. The reasons for preferring Lead CR (and IV R) to Lead CF in cases of pericarditis are given.

5. For routine work, when only one precordial lead is taken, we believe that Lead IV R (apex and right arm) is the one of choice, *provided that the heart is not appreciably enlarged.* With *moderate or marked cardiac enlargement,* it seems preferable to place the precordial electrode *medial to the apex* and never farther to the left than the anterior axillary line.

We are indebted to Dr. David L. Farley and to Dr. Garfield G. Duncan for permission to study patients on their wards.

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THE CARDIAC OUTPUT IN COMPENSATION AND DECOMPENSATION IN THE SAME INDIVIDUAL*

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THERE are conflicting reports concerning the level of cardiac output during compensation and decompensation in the same individual. The majority of studies during compensation and decompensation have consisted of two measurements of cardiac output—one during decompensation, the other after improvement. In this study serial determinations of the cardiac output have been made in four patients during various stages of congestive failure. In one patient the output of the heart was measured on two occasions—shortly after the onset of congestive heart failure, and several times following restoration of compensation. In three other patients, who never regained compensation completely, the cardiac output was measured during relative improvement and relapse.

Employing older methods for cardiac output determinations, the accuracy of which when pulmonary congestion is present has been questioned,[1] Meakins and Davies[2] reported increased cardiac outputs after restoration of compensation. In one patient Dautrebande[3] found that the cardiac output decreased as congestive failure appeared. Bansi and Groscurth[4] studied two patients with decompensation, in one of whom the output was larger, and in the other smaller, than normal. Kroetz[5] studied sixteen patients during cardiac decompensation and later during compensation. In this series the average cardiac output during decompensation was 2.08 liters, and after recovery, 3.14 liters. Eppinger, von Papp and Schwarz[6] reported a distinct tendency toward *increased* cardiac output during failure.

With a newer and more reliable method for the measurement of cardiac output during decompensation—the three-sample acetylene method of Grollman—Harrison, Friedman, Clark, and Resnik[7] determined the cardiac output in fifteen patients during, and after recovery from, congestive failure. In only three instances was restoration of compensation associated with an increase of cardiac output. Using their dye-injection method, Kinsman and Moore[8] have found an average increase of 25 per cent in cardiac output following return of compensation in a series of sixteen patients. However, in five of these patients compensation was associated with a slight decrease in cardiac output.

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Our studies in various stages of cardiac failure in the same patients were carried out as follows.

METHOD

All patients were studied under basal conditions. The three-sample acetylene method of Grollman, as modified by Grollman, Friedman, Clark, and Harrison[9] for the measurement of cardiac output in the presence of pulmonary congestion, was employed in all cases. The vital capacity, circulation time, and venous pressure were also determined on the same day. The vital capacity was measured according to the standard established by Blumgart and Weiss.[10] The arm-to-carotid-sinus circulation time was done by the cyanide method of Robb and Weiss.[11] Venous pressure in the cubital vein was determined by the direct method of Moritz and Tabora.[12] Fluoroscopic examination of the heart was made in all cases with especial reference to the amplitude of excursion of the cardiac borders.

RESULTS

The results of the study of the four patients are summarized in the case reports and in the figures and tables.

Comparative studies with the acetylene and direct Fick methods showed close parallelism of results in decompensated patients under basal conditions, although the absolute values were not identical.

We believe, therefore, that the results to be described are significant, but realize that probably no indirect method of measuring cardiac output is accurate to the cubic centimeter.

Case 1, W. P., is of especial interest because of complete restoration of compensation between and following two attacks of congestive failure, and because of the intelligence and cooperation of the patient.

CASE 1.—W. P. *Diagnosis:* Arteriosclerotic and hypertensive heart disease; cardiac hypertrophy and dilatation; congestive heart failure.

A white man, 49 years of age, was admitted to the Cincinnati General Hospital May 26, 1936. The patient complained of slowly increasing dyspnea on exertion for six months and swelling of the ankles for one month. During the two months prior to admission to the hospital he became much worse, with sensations of weakness while driving his automobile and attacks of paroxysmal nocturnal dyspnea. There was no history of substernal pain. Five years earlier he had been told that he had high blood pressure.

Examination revealed a well-developed, well-nourished white man lying flat in bed, not appearing acutely ill. The pupils reacted to light and during accommodation; the retinal arteries were greatly narrowed and compressed the veins which they crossed. The lips were slightly cyanotic. There was some bulging of the chest, posteriorly, at the base. Below the level of the sixth thoracic vertebra the percussion note on the right side was flat, and the intensity of the breath sounds decreased. The heart was generally enlarged. Roentgenographic measurements showed that the shadow of the great vessels was 8 cm. wide at the second rib, and the cardiac shadow 15.5 cm. wide at the fourth rib; the Danzer ratio was 0.62. The rhythm was normal, but the heart sounds were of poor quality. There was a gallop rhythm, and a blowing systolic murmur heard at the apex, transmitted to the left axilla, and diminishing in intensity toward the base. The pulmonic second sound was louder than the aortic second. The blood pressure was 150/120. The liver was not felt, but pitting edema of both ankles was present.

Laboratory Data.—The specific gravity of the urine was 1.010, and a trace of albumin was present. The hemoglobin was 78 per cent, the erythrocyte count 4,100,000, the leucocyte count 7,800, and the differential leucocyte count normal. An electrocardiogram showed left axis deviation; T_1 was inverted, and T_2 and T_3 were isoelectric.

Course.—The usual treatment for cardiac failure was instituted and 700 c.c. of clear fluid were removed from the right chest by thoracentesis. On June 3, 1936, the cardiac output was 3.57 liters per minute, the stroke volume 37 c.c., the vital capacity 2.5 liters, the basal metabolic rate +11, the oxygen consumption 251

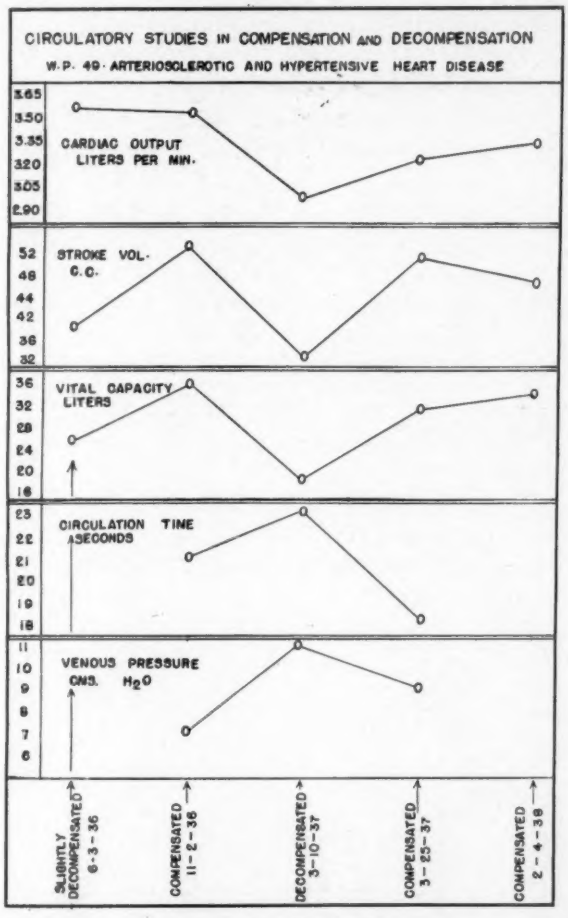

Fig. 1.—Case 1, W. P.

c.c. per minute, and the arteriovenous oxygen difference 70 c.c. per liter. The patient improved sufficiently to be discharged on June 26, 1936. Thereafter he was examined in the Cardiac Clinic at weekly intervals and showed no evidence of congestive failure. Digitalis in doses of 1½ grains daily was continued throughout this period.

On Nov. 2, 1936, when the patient was fully compensated, the cardiac output was 3.56 liters per minute, the stroke volume 52 c.c., the vital capacity 3.5 liters, the circulation time 21 sec., the basal metabolic rate +15, the oxygen consumption 264 c.c. per minute, the arteriovenous oxygen difference 74 c.c. per liter, the venous

pressure 7 cm. of water, and the arterial pressure 164/112. The patient was advised to continue taking digitalis and to return to the Cardiac Clinic at weekly intervals. He did this until Dec. 1, 1936, when he discontinued the digitalis because he was feeling so well. He remained symptom-free for the following three weeks but then began to experience rather severe dyspnea. He resumed digitalis in doses of 1½ grains, but his condition did not improve. The weakness and dyspnea became so marked that he was forced to return to the hospital March 9, 1937.

Examination at this time revealed marked dyspnea but no cyanosis. The neck veins were distended. The heart was enlarged; the apex beat was diffuse and was located in the fifth intercostal space about 11 cm. from the midsternal line.

TABLE I

SUMMARY OF OBSERVATIONS IN CASE 1, W. P.

	ARTERIAL BLOOD PRESSURE (MM.HG)	OXYGEN CONSUMPTION PER MINUTE (C.C.)	CIRCULATION TIME (SEC.)	VITAL CAPACITY (LITERS)	ARTERIOVENOUS OXYGEN DIFFERENCE PER LITER (C.C.)	VENOUS PRESSURE (CM. H_2O)	CARDIAC OUTPUT PER MINUTE (LITERS)	STROKE VOLUME (C.C.)
6/3/36 Slightly decompensated.		251 (B.M.R. +11)		2.5	71.3 69.3		3.57	37
11/2/26 Compensated.	164/112	264 (B.M.R. +15)	21	3.5	75.2	7.0	3.56	52
3/10/37 Decompensated; marked congestion of lungs; dyspnea; feeble cardiac pulsations.	180/140	286 (B.M.R. +20)	23	1.8	94.5 100.0	11.0	2.94	32
3/25/37 Compensated; no evidence of failure; cardiac pulsations more vigorous but still below normal.	160/130	258 (B.M.R. +10)	18	3.1	79.7 81.7	9.0	3.20	50
2/4/38 Compensated; cardiac pulsations full and active.	176/120	288		3.3	85.0 91.0		3.27	45

The teleoroentgenogram disclosed the fact that the size of the cardiopericardial shadow had increased slightly since the previous examination; there was marked congestion of both lungs. The rhythm was normal except for an occasional premature contraction. The heart sounds were of poor quality. There was a high pitched, blowing systolic murmur heard at the apex and transmitted to the axilla. The aortic second sound was louder than the pulmonic second. The blood pressure was 188/140. Numerous râles were heard at the bases of both lungs. The edge of the liver was felt two fingerbreadths below the costal margin and was slightly tender. There was no dependent edema.

Laboratory Data.—The specific gravity of the urine was 1.015; a trace of albumin and acetone and a few hyaline casts were present. The hemoglobin was

15 gm. per cent, the erythrocyte count 5,170,000, the leucocyte count 9,850, and the differential leucocyte count normal. The blood Kahn reaction was negative. The urea nitrogen content of the blood was 15 mg. per cent.

On March 10, 1937, after the usual treatment, the cardiac output was 2.94 liters per minute, the stroke volume 32 c.c., the arteriovenous oxygen difference 97 c.c. per liter, the vital capacity 1.8 liters, the circulation time 23 sec., the basal metabolic rate +20, the oxygen consumption 286 c.c. per minute, and the venous pressure 11 cm. of water. Fluoroscopic examination revealed feeble cardiac pulsations.

After completion of the above studies 2 c.c. of mercupurin were administered intravenously, which resulted promptly in a diuresis of 3000 c.c. Three days later the bases of the lungs were free of congestion, the liver could not be felt, the dyspnea was greatly diminished, and the arterial pressure was 154/120.

On March 25, 1937, when compensation was fully restored, the cardiac output was 3.20 liters per minute, the stroke volume 50 c.c., the arteriovenous oxygen difference 80.7 c.c. per liter, the vital capacity 3.1 liters, the circulation time 18 sec., the basal metabolic rate +10, the oxygen consumption 258 c.c. per minute, and the venous pressure 9 cm. of water.

The patient was discharged March 26, 1937, and advised to continue taking 1½ grains of digitalis daily. Since that time he has returned regularly to the Cardiac Clinic and has had no signs or symptoms of decompensation.

On Feb. 4, 1938, when the patient was symptom-free and actively engaged in his occupation as a salesman, the cardiac output was 3.27 liters per minute, the stroke volume 45 c.c., the arteriovenous oxygen difference 88 c.c. per liter, the vital capacity 3.3 liters, the basal metabolic rate +24, the oxygen consumption 288 c.c. per minute, and the arterial pressure 176/120. Fluoroscopic examination showed active cardiac pulsations.

CASE 2.—J. H. *Diagnosis:* Syphilitic heart disease; aortic insufficiency; congestive heart failure.

A 39-year-old colored man was admitted to the Cincinnati General Hospital May 17, 1937, complaining of shortness of breath and swelling of the abdomen and ankles for the preceding six months. During the five months prior to admission to the hospital he had become progressively worse, with severe attacks of paroxysmal nocturnal dyspnea associated with severe cough which was productive of frothy and frequently blood-tinged sputum. The edema at first involved only the ankles, but gradually spread upward to the legs, thighs, and abdomen. The patient was confined to bed for four months prior to admission to the hospital. He had been told three months earlier that he had "a leak of the heart and bad blood." There was no history of previous cardiac failure.

Examination revealed a well-developed, well-nourished colored man, lying in semi-Fowler's position, appearing acutely ill, suffering from marked dyspnea and orthopnea, and occasionally coughing up frothy, blood-tinged sputum. There was marked edema of the lower extremities and of the trunk to the level of the umbilicus. The neck veins were distended. The pupils reacted to light and during accommodation. The retinal arteries showed moderate tortuosity and narrowing. No cyanosis was present. The respirations were rapid and shallow. There were numerous moist râles in both lungs extending to the angles of the scapulae, with signs of fluid at the right base. The heart was enlarged to the left. The diffuse apex impulse was felt in the sixth intercostal space 11 cm. from the midsternal line. Retrosternal dullness was 7 cm.; relative cardiac dullness 4 by 15 cm. The rhythm was normal. There was a marked gallop rhythm at the apex, and soft blowing systolic and diastolic murmurs were heard at the base. The blood pressure was 145/45. The pulse was of the Corrigan type and Duroziez's sign was present. The edge of the liver was felt three fingerbreadths below the costal margin, and there

was shifting dullness in the flanks. There was marked pitting edema from the costal margins downward. The reflexes were all present and active.

Laboratory Data.—The urine contained a moderate amount of albumin (++); its specific gravity was 1.025. The hemoglobin was 12 gm. per cent, the erythrocyte count 4,200,000, the leucocyte count 9,600, and the differential leucocyte count normal. The blood Kahn reaction was positive (+++). The urea nitrogen content of the blood was 20 mg. per cent. The electrocardiogram showed left axis deviation; T_1 and T_2 were isoelectric, and T_3 diphasic.

Course.—The patient was given a Karell diet and digitalized. Two days later, May 19, 1937, the cardiac output was 1.75 liters per minute, the stroke volume 16 c.c., the arteriovenous oxygen difference 136.5 c.c. per liter, the vital capacity

TABLE II

SUMMARY OF OBSERVATIONS IN CASE 2, J. H.

	ARTERIAL BLOOD PRESSURE (MM.HG)	OXYGEN CONSUMPTION PER MINUTE (C.C.)	CIRCULATION TIME (SEC.)	VITAL CAPACITY (LITERS)	ARTERIOVENOUS OXYGEN DIFFERENCE PER LITER (C.C.)	VENOUS PRESSURE (CM. H_2O)	CARDIAC OUTPUT PER MINUTE (LITERS)	STROKE VOLUME (C.C.)
5/19/37 Decompensated 4+; edema 3+.	150/40	240 (B.M.R. +14)	43	1.22	136.2 137.5	14.5	1.75	16.0
5/20/37 Condition unchanged.	150/50	248 (B.M.R. +18)		1.38	137.0 135.5		1.81	15.3
5/21/37 Considerably improved; some edema; dyspnea.	145/30	232 (B.M.R. +10)	35	1.82	116.2 110.2	5.0	2.10	18.4
6/3/37 No edema; dyspnea on slight effort; failure at bed rest occurred 3 days later.	110/50	202 (B.M.R. −3)	33	1.90	121.0 88.5	5.0	1.98	18.0

1.22 liters, the circulation time 43 sec., the basal metabolic rate +14, the oxygen consumption 240 c.c. per minute, and the venous pressure 14.5 cm. of water.

On the next day, May 20, 1937, the cardiac output was 1.81 liters per minute, the stroke volume 15.3 c.c., the arteriovenous oxygen difference 135.0 c.c. per liter, the vital capacity 1.38 liters, the basal metabolic rate +18, and the oxygen consumption 248 c.c. per minute.

The patient was given 2 c.c. of mercupurin intravenously, which was followed by a prompt diuresis of 6.5 liters with a twenty-four pound loss of weight in twenty-four hours. On May 21, 1937, there were a few râles at the base of the right lung, a protodiastolic gallop rhythm at the apex, and to-and-fro murmurs at the base of the heart. There was much less peripheral edema. On this day the cardiac output was 2.10 liters per minute, the stroke volume 18.4 c.c., the arteriovenous oxygen difference 113.0 c.c. per liter, the vital capacity 1.82 liters, the circulation time 35 sec., the basal metabolic rate +10, the oxygen consumption 232 c.c. per minute, and the venous pressure 5 cm. of water.

The patient continued to improve steadily, and on June 3, 1937, the lungs showed no abnormalities, and the peripheral edema had disappeared. However, gallop rhythm persisted, and there was marked dyspnea on the slightest exertion. On this day the cardiac output was 1.98 liters per minute, the stroke volume 18.0 c.c., the arteriovenous oxygen difference 105.0 c.c. per liter, the vital capacity 1.9 liters, the circulation time 33 sec., the basal metabolic rate –3, the oxygen consumption 202 c.c. per minute, and the venous pressure 5 cm. of water.

Five days later the patient began to have a daily fever (101° to 103° F.), and at absolute bed rest developed frank congestive failure with a return of all

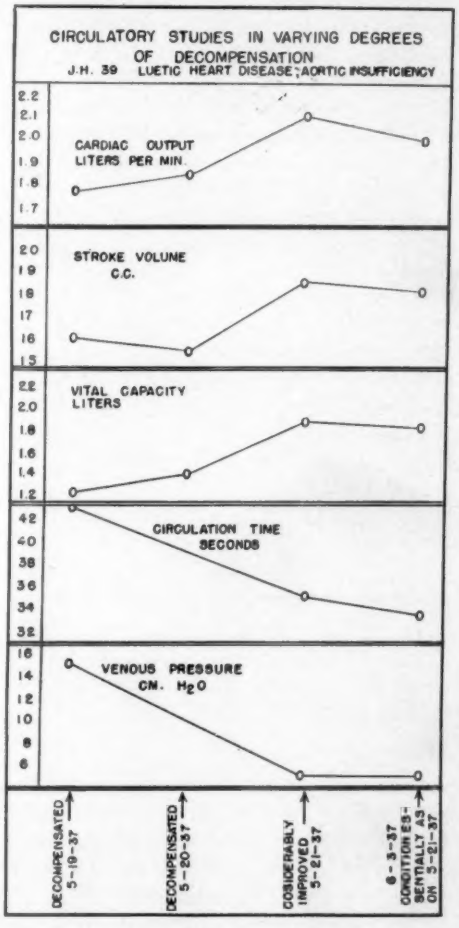

Fig. 2.—Case 2, J. H.

his previous symptoms. However, with the usual therapy, he improved sufficiently to be discharged June 17, 1937. Two months later the patient returned to the hospital and died with congestive heart failure.

CASE 3.—M. B. *Diagnosis:* Arteriosclerotic and hypertensive heart disease; congestive heart failure.

A white woman, 76 years old, was admitted to the Cincinnati General Hospital March 18, 1936, complaining that she had been short of breath for one year and had had swelling of the ankles for three months. Two weeks prior to admission

she began to have frequent and rather severe attacks of paroxysmal nocturnal dyspnea associated with a sensation of strangling. At that time she observed an increase in the edema of her legs and noted that the abdomen was increasing in size. These symptoms became progressively worse. The patient had been told about one year before that she had high blood pressure.

Examination revealed an obese white woman, sitting up in bed, with rather marked dyspnea. There was slight cyanosis of the lips and nail beds. The neck veins were distended. The pupils were irregular and reacted sluggishly to light and during accommodation. Examination of the fundi showed moderate tortuosity of the retinal arteries and compression of the veins which they crossed. The chest was barrel-shaped and the percussion note was slightly impaired at the base of the right lung. There were numerous moist râles at the bases of both lungs. The heart showed general enlargement. Teleoroentgenographic measurements were retrosternal dullness 7.5 cm. at the second rib, 13 cm. at the fourth rib. The greatest cardiac diameter was 18.5 cm. There was marked widening of the aortic arch

TABLE III

SUMMARY OF OBSERVATIONS IN CASE 3, M. B.

	ARTERIAL BLOOD PRESSURE (MM.HG)	OXYGEN CONSUMPTION PER MINUTE (C.C.)	VITAL CAPACITY (LITERS)	ARTERIOVENOUS OXYGEN DIFFERENCE PER LITER (C.C.)	VENOUS PRESSURE (CM. H_2O)	CARDIAC OUTPUT PER MINUTE (LITERS)	STROKE VOLUME (C.C.)
4/28/36 Slightly decompensated.	190/100	221 (B.M.R. +20)	1.58	77.0 87.0	20	3.08	55
2/12/37 Severely decompensated; very dyspneic.	140/100	248	0.85	95.5 93.5	16	2.62	30
2/13/37 Condition unchanged.	155/90	260	0.80	104.0 92.0		2.72	32

and calcification of the aorta. There was also accentuation of the pulmonary conus with enlargement of the right ventricle. The rhythm was normal. There was a blowing systolic murmur heard over the aortic and mitral areas. The blood pressure was 185/120. The tender liver edge was felt three fingerbreadths below the right costal margin. Free fluid was present in the abdominal cavity. There was marked edema of the lower extremities.

Laboratory Data.—The specific gravity of the urine was 1.015, and it contained a trace of albumin. The hemoglobin was 80 per cent, the erythrocyte count 4,400,000, the leucocyte count 8,500, and the differential leucocyte count normal. The blood Wassermann reaction was negative. The urea nitrogen content of the blood was 32 mg. per cent, and the carbon dioxide combining power 61 volumes per cent. The electrocardiogram showed nodal rhythm of type II and left axis deviation; T_1 was inverted, and T_2 and T_3 were diphasic.

Course.—The usual treatment was instituted. After one month of absolute bed rest patient showed slight improvement, but compensation was not restored.

On April 28, 1936, the cardiac output was 3.08 liters per minute, the stroke volume 55 c.c., the venous pressure of 20 cm. of water, the arteriovenous oxygen difference 82 c.c. per liter, the vital capacity 1.6 liters, the basal metabolic rate

+20? (exact estimation of body surface area impossible because of edema), the oxygen consumption 221 c.c. per minute, and the arterial pressure 190/100.

Sixteen days later the patient had improved sufficiently to be discharged from the hospital. She was advised to continue taking digitalis and to return to the Cardiac Clinic, but she did not follow these instructions.

On October 27, 1936, she was readmitted to the hospital with frank congestive failure. After four weeks of hospitalization the edema disappeared, but effort dyspnea persisted. On Nov. 24, 1936, she was discharged from the hospital but advised to remain in bed at her home.

Six weeks later, following an acute upper respiratory infection, the patient again developed congestive heart failure, and was readmitted to the hospital Feb. 11, 1937.

Examination at this time revealed massive edema of the extremities and abdomen. There were marked dyspnea and orthopnea, distention, and pulsation of

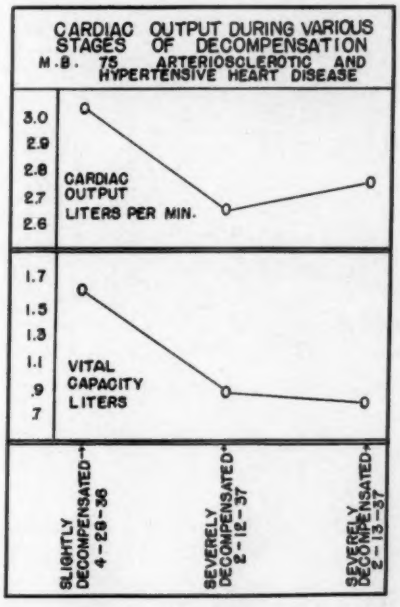

Fig. 3.—Case 3, M. B.

the cervical veins. There were numerous moist râles at the bases of the lungs and signs of small amounts of free fluid in both pleural cavities. The edge of the liver was palpable four fingerbreadths below the right costal margin. The blood pressure was 140/100.

On Feb. 12, 1937, the cardiac output was 2.62 liters per minute, the stroke volume 30 c.c., the venous pressure 16 cm. of water, the arteriovenous oxygen difference 94.5 c.c. per liter, the vital capacity 0.85 liters, and the oxygen consumption 248 c.c. per minute.

The patient was given 2 c.c. of mercupurin intravenously. This was followed by a diuresis of 2,600 c.c. in five hours, with no change in the dyspnea or appreciable effect on the peripheral edema.

Twenty-four hours later the cardiac output was 2.72 liters per minute, the stroke volume 32 c.c., the arteriovenous oxygen difference 98.0 c.c. per liter, the vital capacity 0.80 liters, the oxygen consumption 260 c.c. per minute, and the arterial pressure 155/90.

The patient became progressively worse, and developed intense cyanosis of the lips and nail beds. The edema of the lower extremities, ascites, and hydrothorax remained practically unchanged in spite of a fairly good mercupurin diuresis. Death occurred Feb. 17, 1937.

The anatomical diagnosis was: Generalized arteriosclerosis; cardiac hypertrophy and dilatation, especially right-sided; chronic passive congestion of the viscera; chronic pulmonary emphysema, and arterial nephrosclerosis.

CASE 4.—M. H. *Diagnosis:* Syphilitic and arteriosclerotic heart disease; aortitis with aortic insufficiency and relative mitral insufficiency; congestive heart failure.

A colored man, 41 years old, was admitted to the Cincinnati General Hospital March 2, 1937. He complained of slowly increasing dyspnea on exertion for the preceding year and swelling of the ankles for five months. During the five months prior to admission these symptoms became progressively worse, and were

TABLE IV

SUMMARY OF OBSERVATIONS IN CASE 4, M. H.

	ARTERIAL BLOOD PRESSURE (MM.HG)	OXYGEN CONSUMPTION PER MINUTE (C.C.)	CIRCULATION TIME (SEC.)	VITAL CAPACITY (LITERS)	ARTERIOVENOUS OXYGEN DIFFERENCE PER LITER (C.C.)	VENOUS PRESSURE (CM. H_2O)	CARDIAC OUTPUT PER MINUTE (LITERS)	STROKE VOLUME (C.C.)
3/4/37 Decompensated. Pulmonary and peripheral edema +++, orthopnea.	210/68	435 (B.M.R. +59)		2.3	81.5 86.4	10.0	5.19	51
3/24/37 Discharged. Few rales; no peripheral edema.	150/35	258 (B.M.R. −6)	23	3.7	67.0 74.0	2.5	3.67	46
6/4/37 Decompensated. Edema ++.	170/70	324 (B.M.R. +19)	30	3.1	87.0 85.5	5.0	3.76	38

accompanied by coughing, the expectoration of blood-tinged sputum, and severe attacks of paroxysmal nocturnal dyspnea. Six months prior to admission he was told that he had "high blood pressure and a leaking heart."

The patient had been in the hospital seven years before, when a thyroidectomy was performed for relief of obstructive symptoms in the larynx. He made an uneventful recovery. There was the history of a chancre in 1926, after which he received "shots" twice a week for three months.

The patient was a well-developed and well-nourished colored man suffering from severe dyspnea and orthopnea. Frequent short periods of apnea accompanied by loss of consciousness and dropping of the head were noted. There was moderate distention of the neck veins. The pupils were small, regular, and reacted sluggishly to light and during accommodation. The retinal arteries were narrowed and tortuous and compressed the veins which they crossed; the discs were normal. The percussion note was impaired at the bases of both lungs, where numerous râles were heard. The heart showed general enlargement with diffuse heaving precordial pulsations. The apex beat was diffuse and located in the fifth and sixth intercostal spaces 11 cm. from the midsternal line. The retrosternal dullness was 5 cm., relative

cardiac dullness 3 x 11 cm. The rhythm was normal. High pitched systolic and diastolic murmurs were heard in the third left intercostal space. The blood pressure was 200/70. All of the accessible peripheral vessels were thickened and sclerotic. The arterial pulse was of the Corrigan type. The abdomen was distended, with shifting dullness in the flanks and a definite fluid wave. The edge of the liver was not felt. The lower extremities showed marked pitting edema extending up to the knees. All of the reflexes were present and active.

Laboratory Data.—The specific gravity of the urine was 1.025, and it contained a little albumin (+). The hemoglobin was 13.5 gm. per cent, the erythrocyte count 4,250,000, the leucocyte count 7,700, and the differential leucocyte count

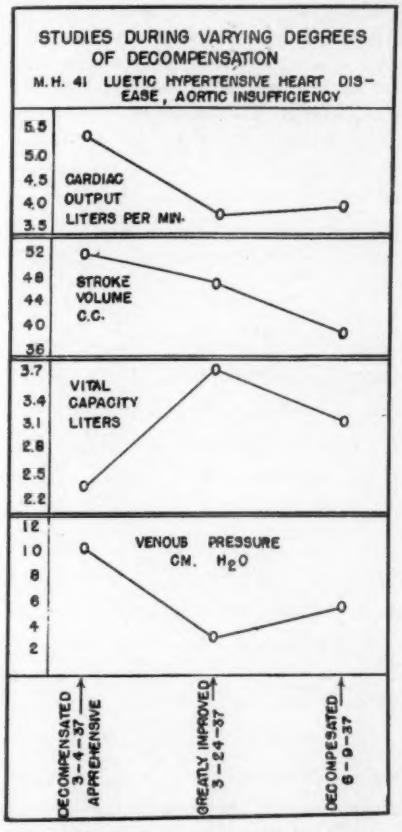

Fig. 4.—Case 4, M. H.

normal. The blood Wassermann reaction (cholesterinized antigen) was rather strongly positive (+++). The phenolsulphonephthalein excretion was 55 per cent in two hours. The electrocardiogram showed left axis deviation, a P-R interval of 0.26 sec., slurring of QRS in Leads I, II, and III, and inversion of T_3.

Course.—The patient was given a Karell diet and digitalized. Two days later, March 3, 1937, the cardiac output was 5.19 liters per minute, the stroke volume 51 c.c., the venous pressure 10 cm. of water, the arteriovenous oxygen difference 83.9 c.c. per liter, the vital capacity 2.3 liters, the basal metabolic rate +59 (patient very apprehensive), the oxygen consumption 435 c.c. per minute, and the arterial pressure 210/68 (patient's weight was 200 pounds; surface area 2.08 square meters).

During the next twenty days the patient improved sufficiently to be discharged from the hospital, at which time the cardiac output was 3.67 liters per minute, the stroke volume 46 c.c., the arteriovenous oxygen difference 70.5 c.c. per liter, the venous pressure 2.5 cm. of water, the vital capacity 3.7 liters, the circulation time 23 sec., the basal metabolic rate −6, and the oxygen consumption 258 c.c. per minute.

The patient was advised to return to the Cardiac Clinic at weekly intervals. During the next two months he was ambulatory, but it was necessary to give him mercupurin twice a week to keep him free of edema.

On June 2, 1937, the patient was readmitted because of congestive failure. There was soft edema of the lower extremities, numerous râles were audible at the bases of both lungs, and the liver was tender and extended three fingerbreadths below the costal margin. There was marked dyspnea on the slightest exertion. The patient was given the usual treatment for heart failure, and two days later, June 4, 1937, the cardiac output was 3.76 liters per minute, the stroke volume 38 c.c., the venous pressure 5 cm. of water, the arteriovenous oxygen difference 86.2 c.c. per liter, the vital capacity 3.1 liters, the circulation time 30 sec., the basal metabolic rate +19, the oxygen consumption 324 c.c. per minute and the arterial pressure 170/70.

The response to treatment was dramatic, and the patient was discharged from the hospital nine days later.

DISCUSSION

In three of the four patients studied, it was found that the more severe the decompensation the lower the cardiac output. In a fourth patient the more severe the failure, the higher the cardiac output.

In a previous report concerning the relationship between cardiac output and cardiac failure,[13] we found that nineteen of twenty patients with congestive failure had subnormal cardiac outputs. Harrison has observed normal, or slightly higher than normal, cardiac outputs not infrequently in decompensated patients, and Kinsman and Moore have occasionally observed normal or supernormal cardiac outputs in the presence of cardiac failure. Thus a subnormal cardiac output is usually, but not invariably, present in cardiac decompensation.

An obvious explanation for the apparent paradox that certain decompensated hearts may maintain a normal output is apparent if one remembers that certain conditions known to increase cardiac output[14] are frequently present in congestive failure, as recently emphasized by Altschule.[15]

1. Exercise (associated with hyperpnea)
2. Increased metabolism
3. Increased venous pressure
4. Long standing anoxemia of tissues
5. Low grade fever
6. Anxiety and apprehension

It is our opinion that the majority of patients with heart failure are not studied under truly basal conditions even though they are con-

tinuously in bed and have fasted for twelve hours. Therefore, it is surprising that the majority of decompensated patients have such low cardiac outputs; the fact that they do suggests a relative inability on the part of the decompensated heart to increase its output in a normal manner.[16]

SUMMARY AND CONCLUSIONS

Repeated determinations of cardiac output have been made in four patients during varying degrees of cardiac decompensation.

In one patient the cardiac output decreased with the onset of congestive heart failure and rose after restoration of compensation.

In two patients, the more severe the decompensation, the lower the cardiac output.

In the fourth patient, the more severe the decompensation, the higher the cardiac output. A theoretical explanation for the apparent paradox has been advanced.

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THE EFFECTS OF THE INTRAVENOUS ADMINISTRATION OF DIGITALIS BODIES ON PATIENTS WITH TRANSIENT VENTRICULAR FIBRILLATION*

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THE purpose of this study was to determine the effects of digitalis bodies on patients with auriculoventricular dissociation subject to transient seizures of ventricular fibrillation. Since digitalis bodies have been found useful in abolishing both single and multiple premature beats of the ventricles[1, 2, 3] in certain forms of cardiac diseases, it was felt that they might be of value in preventing and abolishing the various ventricular irregularities that precede the paroxysms of ventricular fibrillation in patients with either the transient or established forms of auriculoventricular dissociation.

METHOD OF STUDY

One patient with transient auriculoventricular dissociation and two patients with established auriculoventricular dissociation form the subjects of this study. One of these patients showed generalized anasarca and signs of advanced congestive heart failure at the time of the experiment. The other two were free from such signs. The natural course of the development of their attacks and the successive changes in the rhythm of their hearts were studied carefully over a period of several years.[4, 5, 6] During this entire period they were in the Montefiore Hospital, and hundreds of observations made on their heart rhythms were correlated with electrocardiograms.

These experiments were carried out at a time when it was certain that the patients had not had any changes in their cardiac mechanism for at least forty-eight hours. It was definitely determined from study of both the heart and pulse rates, while the patients were connected to the electrocardiographic circuit, that the basic ventricular rate was fairly constant prior to the onset of the experiments, i. e., that it did not vary more than five beats per minute. When the basic rhythm was interrupted spontaneously by premature ventricular beats, the number of such extrasystoles was counted each minute for at least ten minutes prior to the use of any form of digitalis. The patients were kept in bed constantly, and no drug other than a digitalis body was administered to them throughout the entire period of these studies.

On several occasions, frequently before the drug was used, the effects of the intravenous injection of 1 c.c. of either distilled water or physiologic salt solution were determined in order to rule out any abnormal changes in the rhythm of the heart or in the complexes of the electrocardiograms that might follow the injection of the fluid itself.

One of us timed the clinical manifestations following the injection of the drugs, while the other recorded the time intervals at which changes appeared in the electrocardiograms. All studies were carried out with Lead II only. Successive changes in the rhythm of the heart were recorded as frequently as was thought

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necessary, and the movement of the galvanometer string was followed for several hours after the use of the drug unless the condition of the patient made this impossible, in which case reliance was placed upon the clinical manifestations.

Digifolin and ouabain were the drugs used. The minimum dose capable of producing transient changes in the rhythm of the heart was arrived at by the method of trial and error. Starting with very minute quantities, the dose was gradually increased until an effect was produced. The amount finally used was based on the average amount required to give a specific effect.

Although observations were repeated on numerous occasions on the same patient, we have thought it advisable to describe only some of the typical protocols. Each of them demonstrates some mode of action of the drugs to which we direct particular attention. Before mentioning these effects, however, it is important to call attention to the successive changes in the cardiac mechanism which take place when ventricular fibrillation develops spontaneously in patients with either transient or permanent auriculoventricular dissociation, so that comparisons may be made with alterations in the rhythm of the heart that might be caused by the drug.

THE ALTERATIONS IN THE RHYTHM OF THE HEART PRECEDING TRANSIENT PERIODS OF VENTRICULAR FIBRILLATION

Transient ventricular fibrillation may appear spontaneously in patients who exhibit normally a sinus rhythm. The preliminary disturbances that usher in the fibrillatory process in such patients are of two types. One of these is a para-arrhythmia in which the sinus rhythm is interrupted by impulses originating in another center. It may continue for several hours, and may accelerate the heart rate greatly before the basic mechanism is disrupted by short runs of ventricular fibrillation that herald a major seizure.[4, 5] In a second type the heart rate is as a rule slowed at first in the transition from sinus rhythm to auriculoventricular dissociation. There are the usual blocked auricular beats that precede total dissociation, and later, when the ventricles beat independently of the auricles, there is a further slowing of the heart rate. Finally, before ventricular fibrillation appears, acceleration ensues as a result of the interposition of multiple premature beats.

The alterations in rhythm that precede transient periods of ventricular fibrillation during established auriculoventricular dissociation are brought about by (a) an increase in the basic idioventricular rate, (b) the interposition of premature beats which at first come singly and then in groups and, finally, (c) by ventricular oscillations which, in the final analysis, appear to be short runs of ventricular fibrillation.

The idioventricular rate may at times be accelerated in the prefibrillatory period, and this acceleration may be brought about through a variety of mechanisms that have been described elsewhere.[7] Obviously, a study of the effects of any drug upon the cardiac mechanism in patients with established auriculoventricular dissociation must take into account the inherent variability of the idioventricular pacemaker in such patients at a time when they are free from the symptoms that follow ventricular fibrillation. It should be emphasized that our ob-

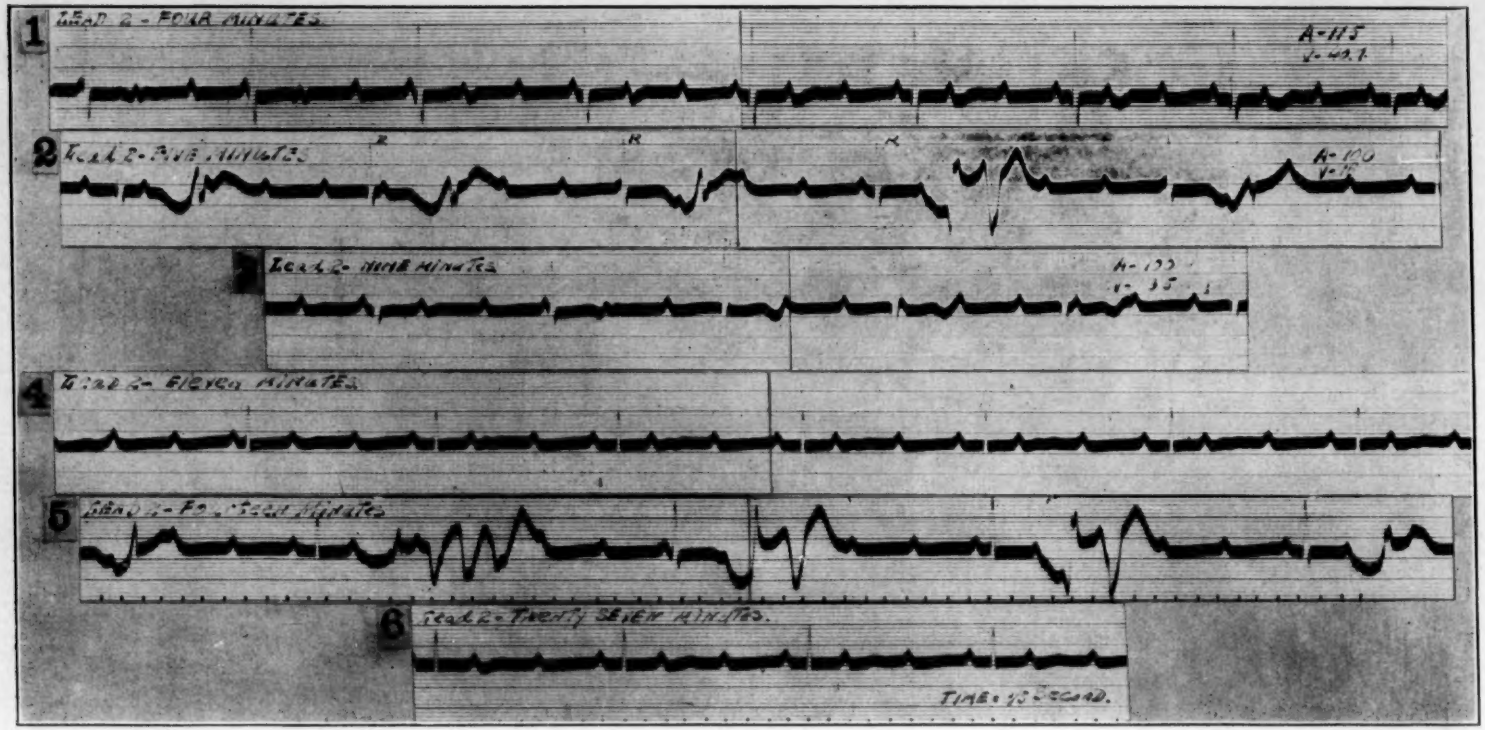

Fig. 1.—A series of electrocardiographic tracings (Lead II only) obtained after the intravenous administration of digifolin to a patient with normal sinus rhythm.

Strip 1: Four minutes after injection of 0.5 c.c. of digifolin. A regular sinus rhythm with a rate of 84 beats per minute is converted to heart block with a ventricular rate of 40.7 and an auricular rate of 115.

Strip 2: Five minutes after the injection of 0.5 c.c. of digifolin. The basic ventricular rate is now 16 beats per minute. The auricles have slowed to 100. Note the interposition of ventricular premature beats.

Strip 3: Nine minutes after the injection of 0.5 c.c. of digifolin. There is a transient return to a heart block with a ventricular rate of 35; the auricular rate is still 100.

Strip 4: The heart block persists for two minutes.

Strip 5: Short runs of ventricular fibrillation begin to disrupt the basic rhythm. Note the markedly aberrant ventricular complexes which follow the basic complexes.

servations were limited to patients in whom some form of dissociation of the auricles from the ventricles was invariably present before the type of fibrillation with which we are concerned set in.

THE EFFECTS OF DIGITALIS BODIES ON A PATIENT WITH NORMAL SINUS RHYTHM WHO DEVELOPED VENTRICULAR FIBRILLATION DURING TRANSIENT AURICULOVENTRICULAR DISSOCIATION

The intravenous administration of 0.5 c.c. of digifolin (the equivalent of ½ cat unit) or of 0.25 mg. ($\frac{1}{240}$ grain) of ouabain was sufficient to change a normal sinus rhythm to partial heart block in a patient who usually showed this transition prior to the development of transient ventricular fibrillation (Fig. 1, strip 1). The same phenomenon was observed in this patient when the experiment was repeated one year later. The injection of 1 c.c. of digifolin yielded the same results. Within four minutes following the administration of either drug there was a sudden change from sinus rhythm with a rate of 90 beats per minute to partial auriculoventricular dissociation with a ventricular rate of 40.7 and an A-V ratio of 3:1. The form of the basic ventricular complexes indicated that the impulse which gave rise to them was supraventricular in origin and that the main ventricular deflections were diphasic. The average R-T segment measured 0.36 sec. and the T-waves (when not masked by a superimposed auricular contraction) were only slightly negative.

One minute later the basic ventricular rate fell to 16 beats per minute (average) and the auricular rate slowed to 100. The ventricular complexes were now diminished in height; the R-T segment was considerably prolonged; and the T-waves assumed a pronounced negativity and were distorted by the superimposed auricular complexes, as well as by the bizarre single and coupled (Fig. 1, strip 2) premature beats of the ventricles that began to disrupt the auriculoventricular dissociation.

These bizarre complexes were in every respect similar to those observed in this patient prior to the spontaneous development of her transient ventricular fibrillation.

During the next six minutes the ventricular beating became regular, and the rate increased to an average of 35 per minute before another paroxysm of ventricular fibrillation occurred (Fig. 1, strips 3 and 4). Clinically, these aberrant ventricular oscillations were frustrate, and consequently they produced no audible sound at the apical region of the heart or perceptible pulse (Fig. 1, strip 5).

On another occasion, fourteen and a half minutes at one time, and twenty minutes at another, after the injection of these drugs, the recurrent ventricular oscillations increased in frequency and duration, so that the intersphygmic intervals became longer. As a result, the patient shut her eyes momentarily, and her face assumed a deathly pallor from the ineffectual distribution of blood to the periphery.

On two separate occasions, twenty-seven minutes and nineteen minutes, respectively, after the administration of the drugs, there were attacks of complete unconsciousness, associated with ventricular fibrillation, which lasted twenty-five and twelve seconds, respectively.

Throughout the rest of the day on which these experiments were carried out the cardiac mechanism was constantly being interrupted by short runs of ventricular fibrillation, a phenomenon that was observed to follow the use of other drugs in such patients.[8, 9] On two such occasions complete restoration of normal sinus rhythm required three or four days.

In this patient no attempt was made to introduce the drugs into the circulation when any abnormal rhythms were present, since at this stage of the patient's illness (she was in the hospital for four and a half years) she invariably developed severe congestive heart failure after having short runs of ventricular fibrillation.

THE EFFECTS OF DIGITALIS BODIES ON PATIENTS WITH ESTABLISHED AURICULOVENTRICULAR DISSOCIATION WHO SHOWED RECURRENT SEIZURES OF TRANSIENT VENTRICULAR FIBRILLATION

The response of patients with auriculoventricular dissociation to the intravenous administration of digifolin and ouabain in the doses described above was variable from time to time, and in the same patient the irregularities induced by the drugs appeared at variable intervals. The onset of the preliminary abnormal rhythms that led to ventricular fibrillation, such as premature beats of the ventricles and short runs of ventricular oscillations, invariably coincided with a slowing of the auricular rate, indicating that these disturbances were caused by the same factor, namely, the administration of digitalis bodies, and were not spontaneous. The time of the appearance of the abnormal rhythms after the injection of the drugs averaged from fourteen to twenty-one minutes. As in the previous patient, once these abnormalities began to appear, they facilitated the development of short runs of ventricular fibrillation which increased in duration and frequency as the day passed and were accompanied at times by periods of unconsciousness when the duration of the intersphygmic intervals exceeded 8 to 20 sec.

Repeated attempts were made to study the effects of the drugs at times when both spontaneous ventricular premature beats and auriculoventricular dissociation were present, but, unfortunately, no correlation could be established between the drug effect and the development of the abnormal mechanism. Since premature beats in themselves very likely facilitate the occurrence of ventricular fibrillation in such patients, with a variable time interval between their inception and that of the fibrillatory process, a correlation could not be established. Suffice it to say that digitalis bodies when injected intravenously did not abolish these abnormal mechanisms at any time.

DISCUSSION

These observations reveal that in patients who are subject to transient seizures of ventricular fibrillation *small* doses of either digifolin or ouabain, administered intravenously, are able to initiate the abnormal mechanism. That the dose need not be large is also indicated by previous observations that ventricular fibrillation may follow the intravenous use of digitalis preparations. For example, von Hoesslin[10] gave only 0.4 mg. of strophanthin K intravenously to a patient who ten minutes later complained of light pains in the precordial region. The electrocardiogram obtained at this time revealed normal rhythm with a ventricular rate of 142 beats per minute. Twenty-five minutes later ventricular fibrillation set in, and ten minutes after that the heart showed no signs of any electrical activity. Von Hoesslin emphasized the fact that the dose need not be large and that fibrillation of the ventricles may begin as late as twenty to thirty minutes after the injection. Similarly, Penati[11] recorded a ventricular tachycardia of 165 to 170 beats per minute in a 42-year-old woman with auricular fibrillation and ventricular extrasystoles who received only 1.8 mg. of strophanthin intravenously. The sudden death of this patient was in all probability due to ventricular fibrillation, for electrocardiograms obtained while she was dying revealed this mechanism. However, attention should be called here to the fact that the tachycardia which is a manifestation of digitalis intoxication need not end in ventricular fibrillation, as is commonly believed.[12] Experiences at the Montefiore Hospital lead us to conclude that in such cases the cause of death is as likely to be ventricular standstill as ventricular fibrillation.[13]

It is very obvious from a study of the natural course of patients who are subject to transient seizures of ventricular fibrillation that some form of block is an essential factor in the attacks. Yet in patients with "block" whose advanced ventricular arrhythmias do not lead to such attacks, the amounts of digitalis used in our experiments never produced ventricular fibrillation. Indeed, it would appear from previous studies of the effect of digitalis on patients with complete heart block that the drug must be used in more than the ordinary therapeutic dose in order to precipitate such abnormal mechanisms.[14] It is evident that the factors responsible for ventricular irregularities in patients with transient seizures of ventricular fibrillation after the injection of small doses of digitalis are as yet not understood.

SUMMARY AND CONCLUSIONS

1. Digifolin and ouabain were administered intravenously to three patients who were subject to transient seizures of ventricular fibrillation, at a time when the basic cardiac mechanism was relatively fixed.

2. Small doses of digifolin (0.5 c.c., the equivalent of ½ cat unit) and ouabain (0.25 mg.), when given intravenously to a patient who

usually showed normal sinus rhythm when she was free from attacks, caused partial heart block within four minutes. After the block became complete, short runs of ventricular fibrillation appeared in this patient within fourteen to twenty-five minutes after the injection.

3. The same doses, when administered to two patients with established auriculoventricular dissociation, caused short runs of ventricular fibrillation within fourteen and a half to twenty-seven minutes after injection.

4. The time of the appearance of the advanced ventricular irregularities was very variable, but the average was eighteen minutes.

5. The administration of these drugs at a time when these patients were having isolated premature beats during established auriculoventricular dissociation did not abolish the abnormal mechanism.

6. Since the intravenous injection of digitalis bodies favors the development of transient ventricular fibrillation in patients who are subject to it, the use of the drug is contraindicated in such patients.

7. The factors responsible for the development of ventricular fibrillation in such patients following the administration of digitalis and ouabain are still unknown.

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FACTORS OF ERROR IN BLOOD PRESSURE READINGS*†

A Survey of Methods of Teaching and Interpretation

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THE purpose in the presentation of this problem is to emphasize the need for a universal standardization of the methods used in the measurement of blood pressure. We have made an effort to determine what variations ordinarily occur in the making of blood pressure observations by interns, attending physicians, and postgraduate medical students in a large postgraduate institution; second, to ascertain how this procedure is taught by medical schools; and third, how it is prescribed by other (medical) institutions which are vitally interested in this subject, such as the life insurance companies. No attempt is made to recommend a standardized method of procedure.

Throughout the country, the most widely accepted and practiced method of measuring blood pressure is the auscultatory method described by Korotkoff in 1905, with the use of a rubber cuff, attached either to a mercury or aneroid manometer, and a stethoscope. In a critical study of blood pressure readings as recorded in various hospital charts by different attending and house physicians, many discrepancies were found. In fact, there was greater variation than the regular limits of error would allow. This latter finding provided the stimulus for this investigation.

It was decided to test the methods employed in taking blood pressure readings by various members of a large general hospital. The New York Post-Graduate Hospital was considered suitable because it is abundantly supplied with physicians from all parts of the world and from many different medical schools. It was decided to make the experiment among three groups: (1) interns, (2) postgraduate students, and (3) attending physicians.

TECHNIQUE

Experiment I. To obviate some of the sources of error a multiaural stethoscope was used so that four different observers could listen to the sounds at the same time on the same patient. The diaphragm of the stethoscope was always placed over the previously palpated brachial

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artery. Effort and attention were concentrated mainly upon the interpretation of the systolic and diastolic phases.

Experiment II. In this experiment an effort was made to record the readings of a large number of observers making blood pressure measurements simultaneously on the same patient. The Cambridge amplifying stethoscope was used. The dial of a Tycos desk manometer was projected on a screen. The cuff was then placed on the arm of the patient and inflated. The stethoscope was placed over the previously palpated brachial artery.

Cards were filled out by the observers, stating from what medical school they graduated, the year of their graduation and their present affiliations, the results of their findings, and how they derived the systolic

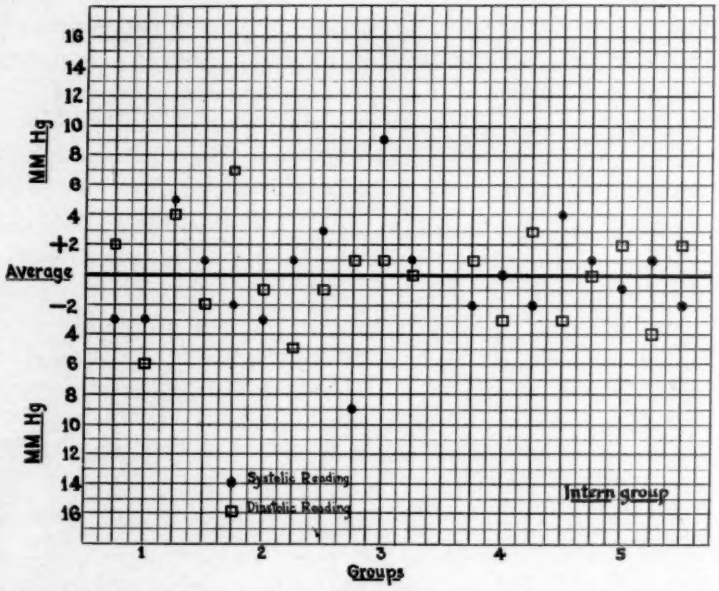

Chart 1.—Experiment 1A. Showing variation in recorded results when four interns at a time took blood pressure readings according to their individual methods of interpretation.

In all charts the average of each set of readings is represented as the "Average" line. The deviations in mm. of Hg for both systolic and diastolic are plotted from this.

and diatolic readings. They were also asked not to make any comparison or communication concerning their results before recording them.

Charts 1 to 5 indicate in graphic form the deviation from the average in millimeters of mercury.

The patients were not selected for this study. They were chosen at random, but patients suffering with hypertension and auricular fibrillation, as well as patients with apparently normal circulatory systems, were included. In single experiments differences in diastolic readings amounting to as much as 20 mm. of mercury, and in systolic readings to as much as 16 mm. of mercury, were observed.

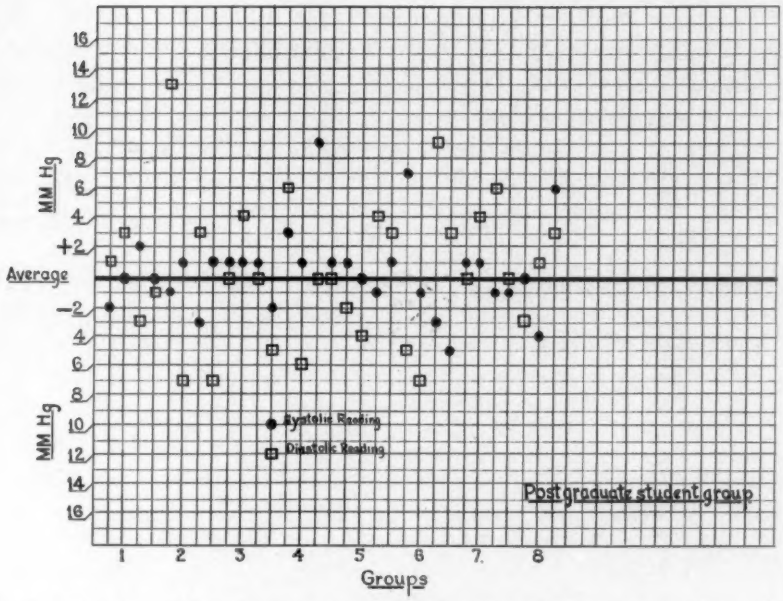

Chart 2.—Experiment 1B. Same as 1A, observations by postgraduate medical students from widely scattered areas.

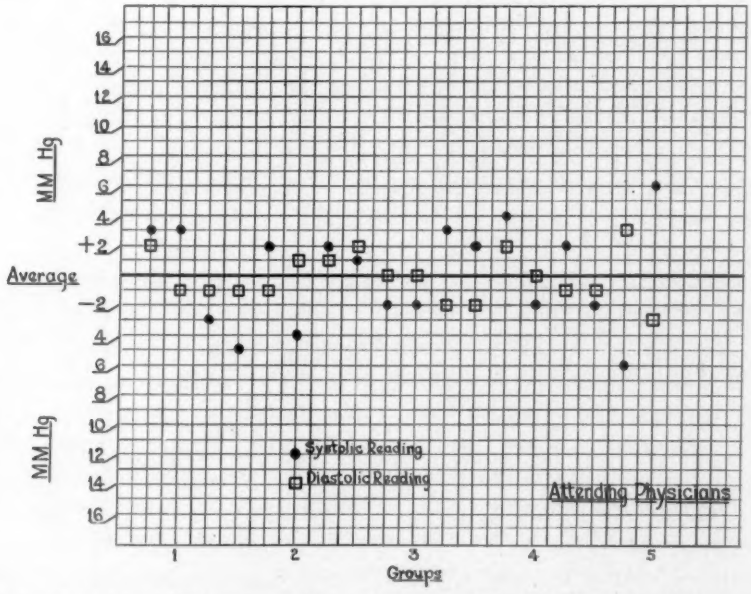

Chart 3.—Experiment 1C. Same as 1A, observations by attending physicians on the Medical Service of the New York Post-Graduate Hospital.

In correlating the methods of determination of the diastolic and systolic phases, which merely meant the interpretation of the sounds as they came through beneath the cuff, the following criteria were used:

Intern Group		
Systolic:	1st sound heard	90%
	1st loud sound heard	10%
Diastolic:	Sudden muffling of sound (4th phase)	90%
	Disappearance of sound (5th phase)	10%
Attending Physician Group:		
Systolic:	1st sound heard	90%
	1st loud sound heard	10%
Diastolic:	Sudden muffling of sound (4th phase)	50%
	Disappearance of sound (5th phase)	50%
Postgraduate Group		
Systolic:	1st sound heard	75%
	1st loud sound heard	25%
Diastolic:	Sudden muffling of sound (4th phase)	52%
	Disappearance of sound (5th phase)	48%

This study not only showed a marked difference in criteria, but also a wide variation in the interpretation of the sounds. Consequently, the next step to be taken was obvious. With the consent and encouragement of the Executive Committee of the American Heart Association, questionnaires were sent to the directors of the medical departments of forty of the leading medical schools of this continent, in an attempt to find out whether there exists a standard method of measuring blood pressures for all the schools and within each school. To date, twenty-four answers have been received.

If the results obtained by the use of the four-way stethoscope were surprising, the replies from the medical schools were even more so, and in a way explained the variations which we had observed.

Of the twenty-four replies, in 67 per cent it was stated that a standard method was employed and taught in the medical department, and in 33 per cent that no standard method was taught. It should be pointed out that the schools with standard methods varied widely in their opinions as to which methods should be used. Eighty-three and one-third per cent of those replying agreed that the first sound heard marked the systolic pressure. Sixteen and two-thirds per cent taught differently in cases of disordered rhythm and hypertension. The diastolic level was taken in 62.5 per cent as the point where the sounds suddenly become muffled (4th phase). Twelve and one-half per cent taught that the disappearance of the sound (5th phase) marked the diastolic pressure, and 25 per cent

taught that either the 4th or 5th phase could be interpreted as indicating the diastolic level, which would be very confusing. One school taught that the loudest sound was the diastolic point, and another school stated merely that the interpretation of the diastolic pressure was "variable."

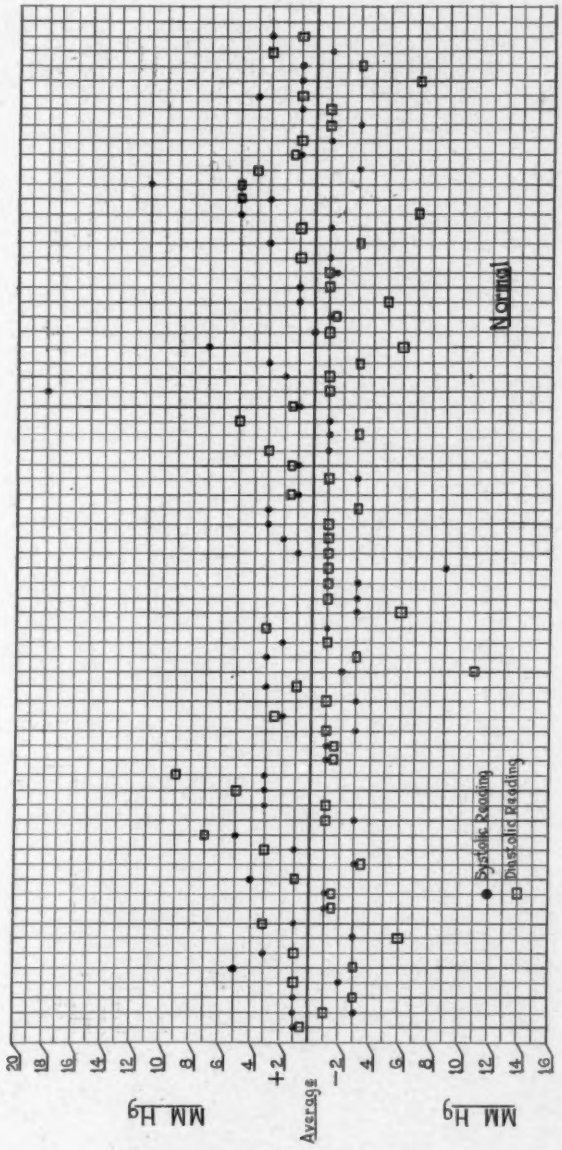

Chart 4.—Experiment 2A. Simultaneous observations made by 68 physicians on a normal subject by means of a loud-speaker.

Further investigation has established the fact that some schools which claim to teach a standard method in practice actually do not. To quote a professor of a leading medical school: "At the same time I will make this prophecy, that the replies which you will get from those responsible for the clinical teaching in the universities will be a poor index of what

is actually taught. The replies will indicate that certain techniques are taught, but in actual practice each clinician follows his own preconceptions as to what is proper."

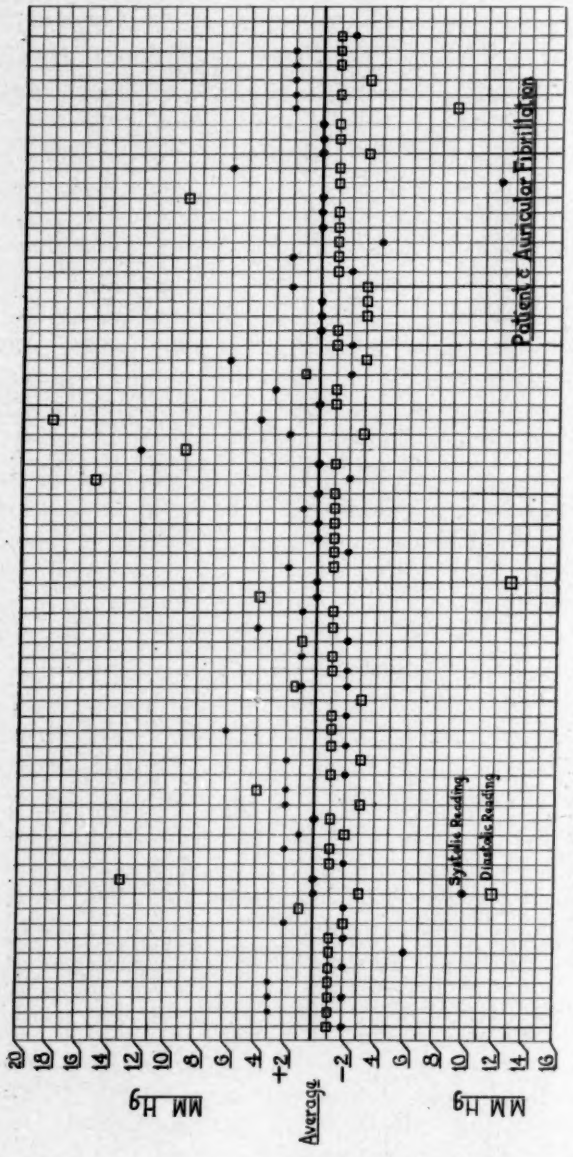

Chart 5.—Experiment 2B. Same as 2A. Patient with auricular fibrillation.

Other factors of error have been investigated by many clinicians. Erlanger,[1] in 1904, experimented with cuffs of different widths, varying from 5 to 17 cm. He found the error progressively less with increasing width of the cuff, but it did not seem to be entirely eliminated even with the 17 cm. cuff. In routine use a 12 cm. cuff was found to present the

smallest degree of error and was accepted as standard, since any cuff of greater width was found to be impractical. Hensen,[2] Janeway,[3] and Hill and Flack[4] showed that, in the absence of edema and muscular contraction and with a sufficiently wide cuff, the size of the arm makes little difference in the reading. Kilgore,[5] in 1918, pointed out the relative merits of the palpatory and auscultatory methods for determining the systolic pressure. Erlanger[1] showed that systolic readings taken while slowly inflating the cuff are usually a little higher than those obtained with falling pressure. Time does not permit a review of the volumes of literature written on the sources of error in blood pressure determinations. Despite these volumes, however, there is no agreement in medical schools today as to what methods should be taught or used clinically or in research.

This problem is, of course, of vital interest and importance to the life insurance companies. A discrepancy of 16 mm. of mercury in the systolic readings and 20 mm. in the diastolic readings, which occurred in one of our experiments, would place an applicant in one of three categories: (1) So-called standard group, with the payment of the ordinary premium; (2) substandard, with an increased premium; or (3) outright declination. Mortality statistics compiled by actuaries have shown that when the systolic pressure is 10 mm. Hg above the average the attained mortality exceeds the expected mortality, and that the percentage of this increase in the attained mortality rises with every increase of 5 mm. in the systolic pressure. This is also true of the diastolic pressure and when both exceed the average the mortality is even greater.

A questionnaire modified from the one used for medical schools was sent to the medical directors of one hundred insurance companies; eighty-two responded, and the replies show that the insurance companies are instructing their examiners differently, as follows:

Of the eighty-two medical directors who replied, thirty-seven, or 45 per cent, stated that they employed a standard method, whereas forty-five, or 55 per cent, declared that their examiners used various criteria. Again the standard methods used varied widely. The systolic pressure was designated by forty-nine companies as the level at which the first sound is heard during deflation of the cuff, by six as that at which the last sound is heard during inflation of the cuff, and by two as that at which the sounds are loudest during deflation. Twenty-five companies confessed that they had no idea how their examiners measured systolic pressure.

The diastolic pressure was designated by forty-eight companies as the point at which the sounds disappear (5th phase), and by six as the 4th phase in cases in which the sounds become muffled suddenly. Sixteen companies required their examiners to report the pressure at both the 4th and 5th phases, and twelve did not know how their examiners were measuring diastolic pressure. The confusion here is no worse than that which exists in the medical schools. Many companies took occasion to

emphasize the fact that the blood pressure is variable when the pulse is irregular; several insisted that the auscultatory method be checked by palpation. Several respondents in the latter group, however, indicated that the blood pressure observations should be made as the arm is being compressed, rather than decompressed.

An interesting commentary was made by three of the companies that their older examiners made the most errors in measuring blood pressure.

That there are marked discrepancies in the making and interpreting of blood pressure observations is beyond dispute. This fact has been known for a long time. Cook,[6] reporting to the Life Insurance Medical Directors Association in 1921, stated that not only were the insurance companies instructing their examiners differently, but that teachers of medicine were not in agreement as to the best methods of measuring blood pressure. He recommended that the Medical Directors Association address a communication to the Association of American Medical Colleges reciting the confusion arising from this failure to standardize such an important clinical procedure and requesting their comments on the subject. We can find no evidence to show that the Medical Colleges took any action at that or any other time.

Clearly, then, this is a problem for the teaching institutions. Individuals cannot be standardized, but procedures for observing their biological processes can be, within certain limits. Important basic biologic measurements should be uniform whenever possible, and they should be so taught in the medical colleges and universities. The making of blood pressure readings is one of the most common procedures in medical practice and research. In the United States and Canada essentially the same equipment is utilized by practically all physicians. The great differences in results as above demonstrated arise for the most part from the confusion as to what is the proper method of measuring blood pressure. The solution is simple providing certain steps are taken. We respectfully submit that the proper officers of the American Heart Association consider the appointment of a National Committee to study this problem for the purpose of taking the steps necessary to provide a standard method of measuring blood pressure for the use of teachers and practitioners of medicine.

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THE EFFECT OF VALVULAR HEART DISEASE ON THE DYNAMICS OF THE CIRCULATION

Observations Before, During and After the Occurrence of Heart Failure*

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IT IS common experience that organic valve defects frequently lead to heart failure, and it has been found that, when this has occurred, the volume output of blood from the heart is decreased.[1, 2] The effects of the valve lesions on the circulation before the onset of failure, however, have not been sufficiently studied. This was apparent when Grollman[3] reviewed the data available in 1932. Starr and his coworkers[4] have made measurements of output in 7 patients with valvular defects before the onset of failure, and Stewart and his associates[5] have also made a few such measurements, but there has been no study of a large series of cases. We have, therefore, made certain measurements of the circulation in 40 patients exhibiting the organic valvular lesions commonly encountered. None had experienced congestive heart failure or was taking drugs. Normal sinus rhythm was present in all. In one (H. W.) the etiology was syphilis, in a second (J. F.) the defect was congenital (coarctation of the aorta), and in the remaining 38 the lesions were those associated with rheumatic infection (Table I). In our analysis, stenosis of a valve is recorded as a lesion and insufficiency as another, so that when both stenosis and insufficiency were present, the patient was said to have two lesions. Five of the patients had single valve lesions, 2 had aortic stenosis and aortic insufficiency, and one had coarctation of the aorta; of the remaining 32, 11 suffered from mitral stenosis and mitral insufficiency, 9 from mitral stenosis and mitral insufficiency and aortic insufficiency, and 12 from mitral stenosis and mitral insufficiency and aortic stenosis and aortic insufficiency (Table I).‡ Only those cases were studied in which the diagnosis of the valve lesion was unequivocal.

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‡In the text of the paper M. I. = mitral insufficiency; M. S. = mitral stenosis; A. I. = aortic insufficiency; A. S. = aortic stenosis; M. S., M. I. = mitral stenosis and mitral insufficiency, etc.

In order to detect differences in functional capacities in progressive heart disease, we have also compared the mitral stenosis and insufficiency group before the onset of failure with a group having the same valve defect after recovery from failure, and with another during congestive heart failure.

We have accumulated the following data under basal conditions: cardiac output, basal metabolic rate, heart rate, height and weight, circulation time, venous pressure, cardiac size in teleoroentgenograms, arterial pressure, physical signs and electrocardiogram.

Thirteen normal individuals were used as controls. A complete report of the observations made in this group will be published elsewhere.[6]

METHODS

All observations were made in the morning while the patients were in a basal metabolic state. All were admitted to the hospital for study. Measurements of the cardiac output* were made by the acetylene method, three samples of gas being

TABLE I

DISTRIBUTION OF CASES

Coaretation of aorta (congenital defect)		1	
Aortic insufficiency (1 rheumatic, 1 syphilitic)		2	
Aortic stenosis	Rheumatic or probably rheumatic	1	
Aortic stenosis and insufficiency		2	
Mitral insufficiency		1	
Mitral stenosis		1	
			8
Mitral stenosis and insufficiency		11	
Mitral stenosis and insufficiency and aortic insufficiency		9	
Mitral stenosis and insufficiency and aortic stenosis and insufficiency		12	
			32
Total			40
Normal individuals			13

taken as recommended by Grollman[3] and by Grollman, Friedman, Clark, and Harrison.[7] During the measurement the patients sat in a steamer chair (angle 135 degrees). They had been made familiar with, and trained to carry out, the procedures beforehand. While they were resting quietly, the cardiac rate was counted at intervals of five minutes. At the end of one-half hour the acetylene-air-oxygen mixture was rebreathed. Three samples of gas were taken during each rebreathing period for estimation of the arteriovenous oxygen difference. The first sample was taken after rebreathing ten to twelve times in 20 seconds, the second after two or three breaths more, and the third after two or three additional breaths. All three samples were usually obtained within 30 seconds. Samples were taken during expiration. The rebreathing procedure was carried out two or three times on each patient. Shortly afterward, the oxygen consumption was measured with a Benedict-Roth

*It is obvious that with this method of measuring the output of the heart in the presence of valve lesions the amount of "regurgitant" blood, which is not circulated through the lungs, cannot be estimated.

spirometer. After a short pause, the vital capacity was measured and height and weight recorded. In succession, sufficient time being allowed in the intervals for the patient to return to a basal metabolic state, an electrocardiogram was taken, the arm-to-tongue circulation time recorded, the venous pressure estimated, and the arterial pressure measured; finally, a roentgenogram of the heart was made at a distance of 2 m.

The arm-to-tongue circulation time was estimated by the use of decholin;[8] 5 c.c. of a 20 per cent solution were injected rapidly (1 to 2 seconds) through an 18 gauge needle into an antecubital vein while the patient was lying quietly in the supine position. This was repeated in one and one-half minutes after the response to the first test had been elicited. The time was recorded from the beginning of the injection until the patient perceived the bitter taste, since a minimal amount of the drug may give a response.

The venous pressure was measured by the direct method,[9] using a large antecubital vein, with the arm at the level of the right auricle. Normal pressures by this method range from 4 to 10 cm. of saline. The antecubital vein of one arm was reserved for the injection of decholin and of the other arm for the measurement of venous pressure. In subsequent measurements the vein was entered at the site first punctured.

Roentgenograms of the heart were taken with the patient in the standing position, during full inspiration, at a distance of 2 m.* Measurements of heart area were carried out by the technique of Levy[10] and estimations of volume were made as recommended by Bardeen.[11]

OBSERVATIONS RELATING TO VALVE DEFECTS BEFORE THE OCCURRENCE OF HEART FAILURE

The data are recorded in Tables II and III, and have also been plotted as frequency diagrams (Fig. 1).

The arteriovenous oxygen difference in the normal individuals averaged 61.4 c.c. (Tables II and III, Fig. 1). It increased to 68.2 c.c. in M. S., M. I., A. I., to 71.2 c.c. in M. S., M. I., and to 78.0 c.c. in M. S., M. I., A. S., A. I. In the few observations relating to the single lesions (to A. S., to A. I., and to M. S.), the arteriovenous oxygen difference did not change significantly; but, on the other hand, A. S., or A. I., or A. S., A. I., when combined with M. S., M. I., increased it.

The average cardiac index was 1.96 liters in M. S., M. I., A. I., 1.86 liters in M. S., M. I., and 1.72 liters in M. S., M. I., A. S., A. I. (Tables II and III, Fig. 1). The normal value is 2.11 liters.

The average stroke volume for the controls was 57 c.c. (Tables II and III, Fig. 1). It decreased to 47 c.c. in M. S., M. I., A. I., to 44 c.c. in M. S., M. I., and still further to 36 c.c. in M. S., M. I., A. S., A. I.

The average stroke volume per kilogram was 0.82 c.c. for normal individuals, but decreased to 0.75 c.c. in M. S., M. I., A. I., to 0.70 c.c. in M. S., M. I., and further still to 0.64 c.c. in M. S., M. I., A. S., A. I. (Tables II and III, Fig. 1).

*The authors are deeply indebted to the X-Ray Department of the New York Hospital for their cooperation in this investigation.

TABLE II

DATA RELATING TO 40 PATIENTS EXHIBITING VALVE DEFECTS BEFORE THE OCCURRENCE OF HEART FAILURE

NAME, SEX, HISTORY NO., AGE	DATE	HEIGHT (CM.)	WEIGHT (KG.)	BODY SURFACE (SQ.M.)	OXYGEN CONSUMPTION (C.C. PER MIN.)	BASAL METABOLIC RATE (PER CENT)	ARTERIOVENOUS OXYGEN DIFFERENCE (C.C.)	CARDIAC OUTPUT (LITERS PER MIN.)	CARDIAC OUTPUT (LITERS PER SQ. M. PER MIN.)	HEART RATE (PER MIN.)	STROKE VOLUME (C.C.)	CARDIAC AREA (SQ. CM.)	CARDIAC VOLUME (C.C.)	ARTERIAL PRESSURE (M.M. HG)	CIRCULATION TIME (SEC.)	VENOUS PRESSURE (CM.)	VITAL CAPACITY (C.C.)	LEFT VENTRICULAR WORK (GM. M. PER BEAT)	STROKE VOLUME PER KG. (C.C.)	LEFT VENTRICULAR WORK PER BEAT PER KG. (GM. M.)	DIAGNOSIS*
Aortic Insufficiency																					
H. W. ♂	10/15/34	173.0	63.4	1.75	227	0	64.1	3.54	2.02	74	48	–	–	118/50	13.2	–	4100	55.0	0.76	0.87	Syph., A.I., E.H.,
No. 71726	10/16/34	172.5	63.1	1.75	232	+ 2	60.3	3.85	2.20	72	53	137.5	774.0	108/40	13.8	–	4100	53.0	0.86	0.84	Class I
48 years	10/17/34	172.8	63.3	1.75	222	- 2	60.1	3.69	2.10	74	49	138.5	787.1	118/46	12.8	–	4250	55.0	0.77	0.87	
M. S. ♂	3/27/34	168.3	76.2	1.87	243	+ 5	53.6	4.53	2.43	84	54	176.8	1135.3	156/64	–	–	4290	80.8	0.71	1.06	Rh., A.I., marked
No. 35859																					E.H., Class IIA
27 years																					
Aortic Stenosis																					
V. T. ♂	12/ 4/37	168.3	61.2	1.78	255	+11	59.0	4.32	2.54	84	51	141.8	815.1	100/70	16.8	4.9	3500	59.0	0.83	0.96	Rh., A. S., E.H.,
No. 186110																					Class IIA
33 years																					

*In this column the following abbreviations are used:

syph. = syphilis
Rh. = rheumatic fever
cong. = congenital
unk. = unknown
M.S. = mitral stenosis
M.I. = mitral insufficiency
A.S. = aortic stenosis
A.I. = aortic insufficiency
E.H. = enlargement of heart
sl. = slight
mark. = marked
? = questionable
R.I.V.H.B. = right intraventricular heart block
I, IIA, IIB, III, refer to functional classification (Criteria for the Classification and Diagnosis of Heart Disease, ed. 2, New York Tuberculosis and Health Association, New York, 1929).

TABLE II—CONT'D

Coarctation of Aorta																					
J. F. ♂ No. 63890 26 years	3/30/35	179.0	66.5	1.82	315	+24	90.3	3.52	1.93	89	41	274.6	2197.0	Rt. 174/60 Lt. 204/60	16.4	8.7	3250	69.7	0.62	1.04	Congenital, Coarctation of aorta, marked E.H., Class I
Aortic Stenosis and Insufficiency																					
W. M. ♂ No. 126827 47 years	3/30/36	165.0	53.5	1.59	245	+18	86.3	2.84	1.79	74	38	154.5	926.4	124/82	16.2	7.9	3500	53.2	0.71	0.99	Rh., A.S., & A.I., sl. E.H., Class I or IIA
J. M. ♂ No. 180633 32 years	10/ 5/37	162.5	78.0	1.84	309	+23	63.7	4.86	2.64	80	61	210.2	1470.3	118/50	15.6	8.2	3000		0.78	0.89	Rh., A.S., & A.I., E.H., Class I
Mitral Insufficiency																					
B. C. ♀ No. 84880 55 years	1/11/35	165.0	57.9	1.64	187	0	70.1	2.67	1.63	72	38	127.6	696.0	122/88	16.3	6.0	3400	54.3	0.66	0.94	Rh., M.I., Class I
Mitral Stenosis																					
R. D. ♂ No. 48714 32 years	12/11/33 10/30/36	184.0 182.3	61.8 60.4	1.82 1.80	237 234	- 5 - 5	61.4 61.8	3.86 3.79	2.12 2.10	66 70	59 54	121.2 129.6	647.0 714.4	105/60 102/70	- 12.7	- 6.9	4700 4600 69.7	65.6 63.2	0.95 0.89	1.06 1.05	Unk., M.S., Class I
Mitral Stenosis and Insufficiency																					
A. G. ♂ No. 79037 23 years	10/30/34 11/12/34 1/10/35 3/28/35	175.5 176.5 177.2 176.0	59.4 59.4 59.6 61.7	1.73 1.75 1.74 1.74	210 214 243 222	-14 -10 - 2 -10	69.9 69.7 71.9 61.4	3.00 3.06 3.38 3.62	1.74 1.79 1.94 2.10	76 76 74 74	39 40 46 50	144.0 141.9 137.7 136.8	832.1 811.0 781.0 772.2	118/80 118/74 104/70 110/70	16.0 18.5 16.5 15.4	- - 11.0 10.3	4200 4300 4200 4200	53.0 52.0 46.7 61.2	0.66 0.67 0.77 0.81	0.89 0.88 0.78 0.99	Rh., M.S., & M.I., E.H., Class I
W. H. ♂ No. 37716 21 years	2/21/34	173.5	88.9	2.03	240	-13	71.7	3.34	1.65	88	38	114.8	595.0	122/90	-	-	4050	54.7	0.43	0.62	Rh., M.S. & M.I., Class I
M. H. ♀ No. 59670 39 years	3/28/34	155.0	50.7	1.48	170	- 8	63.7	2.67	1.81	80	33	130.9	724.0	108/70	-	-	2690	40.3	0.66	0.79	Rh., M.S. & M.I., sl. E.H., Class IIA

TABLE II—CONT'D

NAME, SEX, HISTORY NO., AGE	DATE	HEIGHT (CM.)	WEIGHT (KG.)	BODY SURFACE (SQ.M.)	OXYGEN CONSUMPTION (C.C. PER MIN.)	BASAL METABOLIC RATE (PER CENT)	ARTERIOVENOUS OXYGEN DIFFERENCE (C.C.)	CARDIAC OUTPUT (LITERS PER MIN.)	CARDIAC OUTPUT (LITERS PER SQ. M. PER MIN.)	HEART RATE (PER MIN.)	STROKE VOLUME (C.C.)	CARDIAC AREA (SQ. CM.)	CARDIAC VOLUME (C.C.)	ARTERIAL PRESSURE (MM. HG)	CIRCULATION TIME (SEC.)	VENOUS PRESSURE (CM.)	VITAL CAPACITY (C.C.)	LEFT VENTRICULAR WORK (GM. M. PER BEAT)	STROKE VOLUME PER KG. (C.C.)	LEFT VENTRICULAR WORK PER BEAT PER KG. (GM. M.)	DIAGNOSIS*
J. M. ♂ No. 79052 23 years	11/ 3/34	166.0	60.5	1.66	229	- 2	81.9	2.97	1.68	72	39	128.2	701.0	110/60	13.2	-	3700	45.1	0.64	0.75	Rh., M.S. & M.I., sl. E.H., Class IIA
W. L. ♂ No. 67123 20 years	9/29/34	171.5	60.3	1.72	220	-11	76.4	2.88	1.67	74	39	108.6	583.0	110/80	-	-	3400	50.4	0.65	0.84	Rh., M.S. & M.I., Class IIA
J. M. ♂ No. 85646 18 years	1/28/35	164.0	52.1	1.56	218	- 7	83.3	2.62	1.68	96	27	111.2	647.0	110/68	11.0	7.5	3500	32.7	0.52	0.63	Unk., M.S. & M.I., Class I
A. M. ♂ No.75530 24 years	9/28/35	178.0	71.0	1.88	203	-21	57.6	3.52	1.90	66	54	137.7	781.2	124/70	13.4	12.3	5000	71.2	0.76	1.00	Unk., M.S. & M.I., ? E.H., Class IIA
F. M. ♂ No. 66492 32 years	11/ 1/34	176.5	66.7	1.80	259	+ 6	81.4	3.18	1.77	70	45	138.2	784.4	122/72	14.0	-	4900	59.4	0.67	0.89	Rh., M.S. & M.I., Class IIA
J. T. ♂ No. 68162 20 years	6/28/34	176.0	71.6	1.86	273	+ 8	82.4	3.32	1.80	74	45	114.7	594.1	115/70	-	-	4900	56.9	0.63	0.79	Unk., M.S. & M.I., ? sl. E.H., Class I

TABLE II—CONT'D

C. W. ♂ No. 121630 23 years	3/25/36	174.3	59.7	1.72	255	+ 6	65.2	3.91	2.27	68	57	122.4	655.0	132/78	14.4	8.6	4200	81.4	0.95	1.36	Rh., M.S. & M.I., ? sl. E.H., Class I
R. T. ♀ No. 124470 29 years	3/14/36	167.0	54.0	1.60	218	+ 7	61.1	3.57	2.23	66	54	111.9	553.3	110/58	9.6	8.0	3000	61.7	1.00	1.10	Rh., M.S. & M.I., ? sl. E.H., Class I or IIA
Mitral Stenosis and Insufficiency; Aortic Insufficiency																				
A. B. ♂ No. 56690 23 years	4/10/34	174.6	63.5	1.77	207	-14	74.9	2.88	1.63	90	32	123.6	667.0	136/88	-	-	4650	48.7	0.50	0.77	Unk., M.S. & M.I., A.I., Class I
H. C. ♂ No. 82461 40 years	6/15/35	179.0	79.5	1.97	249	- 5	52.4	4.73	2.40	60	79	127.1	691.0	108/68	12.8	8.2	4700	94.6	0.99	1.19	Rh., R.I.V.H.B., M.S. & M.I., A.I., marked E.H., Class IIA
A. D. ♂ No. 149574 17 years	11/14/36	167.5	55.3	1.62	218	-13	75.0	2.91	1.80	54	54	119.5	632.0	125/68	16.0	5.4	4000	71.2	0.98	1.29	Unk., M.S. & M.I., A.I., ? sl. E.H., Class I
P. H. ♂ No. 63300 22 years	5/22/34	162.8	55.5	1.59	227	+ 5	64.7	3.51	2.21	70	50	148.3	872.0	122/74	-	-	3400	66.6	0.90	1.20	Rh., M.S. & M.I., A.I., E.H., Class IIA
J. L. ♂ No. 59220 25 years	4/19/34	169.5	58.8	1.68	238	+ 4	77.8	3.06	1.82	100	31	135.7	764.3	100/62	-	-	3940	34.2	0.53	0.58	Unk., M.S. & M.I., A.I., Class I
R. L. ♂ No. 90113 21 years	4/ 6/35	172.5	63.1	1.75	234	- 7	70.9	3.30	1.89	74	45	120.6	647.0	120/64	17.2	10.5	4100	56.0	0.71	0.89	Rh., M.I., M.S., & A.I., E.H., Class I
A. M. ♀ No. 37709 23 years	2/14/35	163.2	67.5	1.73	203	- 8	62.7	3.24	1.87	74	44	97.6	670.0	112/56	11.6	10.1	2900	52.0	0.65	0.77	Rh., M.I., M.S., & A.I., E.H., Class I or IIA

TABLE II—CONT'D

NAME, SEX, HISTORY NO., AGE	DATE	HEIGHT (CM.)	WEIGHT (KG.)	BODY SURFACE (SQ.M.)	OXYGEN CONSUMPTION (C.C. PER MIN.)	BASAL METABOLIC RATE (PER CENT)	ARTERIOVENOUS OXYGEN DIFFERENCE (C.C.)	CARDIAC OUTPUT (LITERS PER MIN.)	CARDIAC OUTPUT (LITERS PER SQ. M. PER MIN.)	HEART RATE (PER MIN.)	STROKE VOLUME (C.C.)	CARDIAC AREA (SQ. CM.)	CARDIAC VOLUME (C.C.)	ARTERIAL PRESSURE (MM. HG)	CIRCULATION TIME (SEC.)	VENOUS PRESSURE (CM.)	VITAL CAPACITY (C.C.)	LEFT VENTRICULAR WORK (GM. M. PER BEAT)	STROKE VOLUME PER KG. (C.C.)	LEFT VENTRICULAR WORK PER BEAT PER KG. (GM. M.)	DIAGNOSIS*
F. R. ♂ No. 47031 26 years	1/26/34	166.0	66.7	1.75	219	- 8	77.5	2.83	1.62	72	40	141.1	809.0	130/64	-	-	4350	52.8	0.60	0.79	Rh., M.S. & M.I., A.I., E.H., Class I
H. W. ♂ No. 44299 29 years	1/ 4/35	174.0	60.4	1.72	238	0	57.9	4.18	2.43	80	52	149.1	779.0	128/60	13.0	8.3	3900	66.5	0.86	1.10	Rh., M.S. & M.I., A.I., sl. E. H., Class I
Mitral Stenosis and Insufficiency; Aortic Stenosis and Insufficiency																					
E. P. ♂ No. 14505 21 years	11/ 8/34	167.0	49.3	1.54	195	-12	71.4	2.73	1.77	80	34	119.2	629.0	122/50	19.5	-	3100	40.0	0.69	0.81	Rh., M.I., M.S., A.I., & A.S., E.H., Class IIA
M. C. ♀ No. 67280 46 years	12/10/34	156.5	47.0	1.44	182	+ 5	77.4	2.35	1.63	66	36	125.8	678.4	128/84	15.6	6.7	2600	52.0	0.77	1.10	Rh., M.I., M.S., A.I., & A.S., E.H., Class IIA
J. F. ♀ No. 31027 25 years	5/18/35	164.0	46.3	1.48	122	0	65 9	1.85	1.25	70	26	121.6	647.0	104/50	16.6	6.8	2800	27.0	0.56	0.58	Unk., M.I., M.S., A.I., & A.S., E.H., Class I

TABLE II—CONT'D

E. C. ♀ No. 33078 19 years	3/18/35	164.0	61.0	1.66	195	- 6	82.8	2.36	1.42	86	27	106.9	551.2	104/10	10.7	8.2	2850	21.0	0.44	0.34	Rh., M.I., M.S., A.I., & A.S., E.H., Class I
J. C. ♂ No. 124678 28 years	12/ 9/37	172.4	79.9	1.88	331	+21	75.5	4.15	2.21	80	52	113.0	580.4	128/30	16.4	7.6	4000	55.9	0.65	0.70	Rh., M.S. & M.I., A.S. & A.I., E.H., Class I
E. F. ♂ No. 67656 29 years	6/22/34	167.8	49.0	1.56	253	+18	84.7	2.99	1.90	94	32	247.6	1882.0	130/30	–	–	3100	34.8	0.65	0.71	Rh., M.S. & M.I., A.S., & A.I., marked E.H., Class IIA
I. G. ♂ No. 43913 43 years	6/27/34	163.0	61.2	1.68	249	+16	79.0	3.15	1.87	93	34	168.6	1055.0	155/35	–	–	3700	43.9	0.56	0.72	Rh., M.S. & M.I., A.S. & A.I., E.H., Class IIA
J. H. ♂ No. 28858 20 years	4/11/34 4/15/34 4/18/34	181.5 181.5 181.5	60 8 61.3 61.0	1.79 1.79 1.79	253 251 234	+ 3 + 3 - 4	80.8 90.0 81.9	3.12 2.80 2.86	1.75 1.56 1.60	80 83 84	39 34 34	180.0 180.0† 180.0†	1178.0 1178.0† 1178.0†	166/50 162/40 152/40	– – –	– – –	4300 4230 4210	57.3 46.7 44.4	0.64 0.55 0.56	0.94 0.76 0.73	Rh., M.S. & M.I., A.S. & A.I., E.H., Class IIA
S. H. ♂ No. 58578 45 years	1/ 3/35	179.5	54.2	1.70	218	- 1	81.5	2.67	1.57	84	32	151.5	899.4	86/72	25.0	10.2	2450	34.4	0.59	0.65	Unk., M.S. & M.I., A.S., & A.I., E.H., Class IIA
H. N. ♂ No. 52501 14 years	2/ 6/34	158.5	42.2	1.39	232	+ 5	86.4	2.68	1.93	94	29	124.1	668.0	138/32	–	–	2970	33.5	0.69	0.79	Rh., M.S. & M.I., A.S. & A.I., ? E.H., Class IIA
J. O. ♂ No. 124373 31 years	3/ 7/36	169.7	54.4	1.62	203	- 8	77.7	2.61	1.61	60	44	170.8	1070.1	96/70	22.9	7.3	2500	49.7	0.81	0.91	Rh., M.S. & M.I., A.S. & A.I., E.H., Class IIA
M. S. ♂ No. 30087 33 years	6/ 6/34	166.5	67.5	1.76	235	- 3	67.8	3.47	1.97	64	54	164.2	1015.0	108/60	–	–	3625	61.7	0.80	0.91	Rh., M.S. & M.I., A.S., & A.I., E.H., Class IIA

†X-rays not repeated on these days.

There was no significant deviation from the average normal venous pressure (10.0 cm.) in the patients with valve lesions (Tables II and III, Fig. 1).

The average arm-to-tongue circulation time in the normal subjects was 14.4 sec. There was a slight increase in patients with aortic stenosis, and in the M. S., M. I., A. S., A. I. group it was prolonged to 18.1 sec. (Tables II and III, Fig. 1).

In normal subjects the average work of the left ventricle per beat per kilogram was 1.04 gm.m. This was decreased to 0.95 gm.m. in M. S., M. I., A. I., to 0.88 gm.m in M. S., M. I., and further still to 0.78 gm.m in M. S., M. I., A. S., A. I. (Tables II and III, Fig. 1).

There was a tendency for the oxygen consumption to be greater in the patients with aortic lesions (Fig. 1).

TABLE III

MEAN VALUES OF MEASUREMENTS OF 13 NORMAL INDIVIDUALS* AND OF SUBJECTS EXHIBITING VALVE LESIONS BEFORE FAILURE (TABLE II)

	ARTERIO-VENOUS OXYGEN DIF-FERENCE (C.C.)	CARDIAC INDEX (LITERS PER MIN.)	STROKE VOLUME (C.C.)	STROKE VOLUME (C.C. PER KG.)	LEFT VENTRIC-ULAR WORK PER BEAT PER KGM. (GM.M.)	VENOUS PRES-SURE (CM. PHYSIO-LOGIC SALINE)	CIRCULA-TION TIME (SEC.)
Normal Individuals							
	61.4	2.11	57	0.82	1.04	10.0	14.4
Valve Lesions							
A.I.	59.5	2.19	51	0.78	0.91	–	13.3
A.S.	61.2	2.54	51	0.83	0.96	4.9	16.8
A.S. A.I.	75.0	2.22	50	0.75	0.94	8.0	15.9
M.I.	70.1	1.63	35	0.66	0.94	6.0	16.3
M.S.	61.6	2.11	56	0.92	1.06	6.9	12.7
M.S. M.I.	71.2	1.86	44	0.70	0.88	9.8	13.2
M.S. M.I. A.I.	68.2	1.96	47	0.75	0.95	8.5	14.1
M.S. M.I. A.S. A.I.	78.0	1.72	36	0.64	0.76	7.8	18.1

*Data from Stewart and Watson.[6]

OBSERVATIONS RELATING TO CHANGES ASSOCIATED WITH HEART FAILURE IN MITRAL STENOSIS AND MITRAL INSUFFICIENCY

Observations on groups of patients with mitral stenosis and insufficiency made before congestive failure had occurred, while it was present, and after recovery from failure, together with the same observations on the normal controls,[6] are summarized in Tables IV and V, and are also presented in the form of frequency diagrams (Fig. 2).

In the group of those who had recovered from heart failure, and in the group of those who had failure at the time of our study, there were patients with auricular fibrillation, as well as others with normal sinus mechanism. The rhythm is indicated in the frequency diagrams.

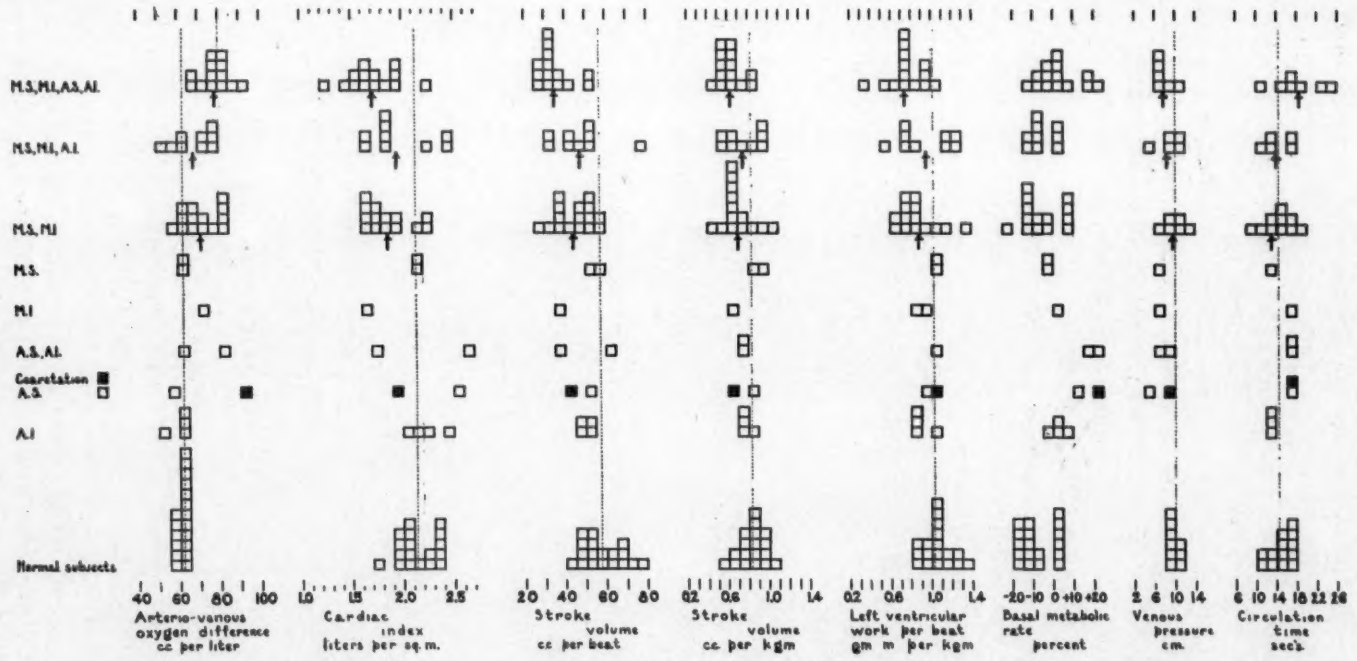

Fig. 1.—In this figure are plotted as frequency diagrams the data relating to measurements of the circulation in the group of normal individuals, and in the valve defect groups (Tables II and III). In this figure, as well as Fig. 2, each square is a unit and represents one measurement, and they are piled on top of one another when there is recurrence of that increment in that valve group. The increments are as follows: arteriovenous oxygen difference, 5 c.c.; cardiac index, 0.1 liter; stroke volume, 5 c.c.; stroke volume per kilogram, 0.1 c.c. per kilogram; left ventricular work per beat per kilogram, 0.1 gm.m.; B.M.R., 5 per cent; venous pressure, 2 cm.; and circulation time, 2 seconds. The means of the normal group have been extended through the other groups by a dotted line. The mean of each value group is indicated by the position of an arrow just below that particular group.

In Table V, averages of both normal sinus rhythm and auricular fibrillation are given, as well as the averages for the two rhythms combined. The combination of the two rhythms does not appear to alter the inferences from the analysis, but separation of the data is possible, if it is desired.

It is recalled that the average normal arteriovenous oxygen difference was 61.4 c.c. (Table V), and that it was increased to 71.2 c.c. in M. S., M. I. before failure. It was increased still further to 79.4 c.c. in those patients who had recovered from failure, and to 99.5 c.c. during heart failure (Tables IV and V, Fig. 2).

The average normal cardiac index amounted to 2.11 liters. In M. S., M. I., before failure, it was 1.86 liters; in those who had recovered from congestive heart failure it was 1.65 liters; and during congestive heart failure it was 1.39 liters (Tables IV and V, Fig. 2).

The average stroke volume amounted to 57 c.c. in normal individuals, but was decreased to 44 c.c. in M. S., M. I. before failure, to 43 c.c. in those who had recovered from congestive heart failure, and to 25 c.c. during failure (Tables IV and V, Fig. 2).

The average stroke volume per kilogram was decreased to 0.70 c.c. in M. S., M. I. before failure (normal, 0.82 c.c.), to 0.76 c.c. in those who had recovered from failure, and to 0.49 c.c. during failure (Tables IV and V, Fig. 2).

The left ventricular work per beat per kilogram was decreased to 0.88 gm.m in M. S., M. I. before failure (normal, 1.04 gm.m), to 0.91 gm.m. in those who had recovered from congestive heart failure, and to 0.68 gm.m. during congestive heart failure (Tables IV and V, Fig. 2).

The average venous pressure was increased to 12.8 cm. in congestive heart failure and was 7.3 cm. after recovery from failure (normal 10.0 cm.) (Tables IV and V, Fig. 2).

The circulation time showed no change from normal (14.4 sec.), in M. S., M. I. before failure (13.2 sec.), but was prolonged to 21.5 sec. in those who had recovered from congestive heart failure and to 27.5 sec. during failure (Tables IV and V, Fig. 2).

DISCUSSION

Analysis of the observations in the 32 cases falling in the M. S., M. I. and M. S., M. I., A. I. and the M. S., M. I., A. S., A. I. groups shows certain definite trends. With respect to arteriovenous oxygen difference, cardiac index, cardiac output per beat, stroke volume per kilogram and left ventricular work per beat per kilogram, the impairment was *least* in M. S., M. I., A. I., greater in M. S., M. I., and greater still in M. S., M. I., A. S., A. I. With respect to all these functions the

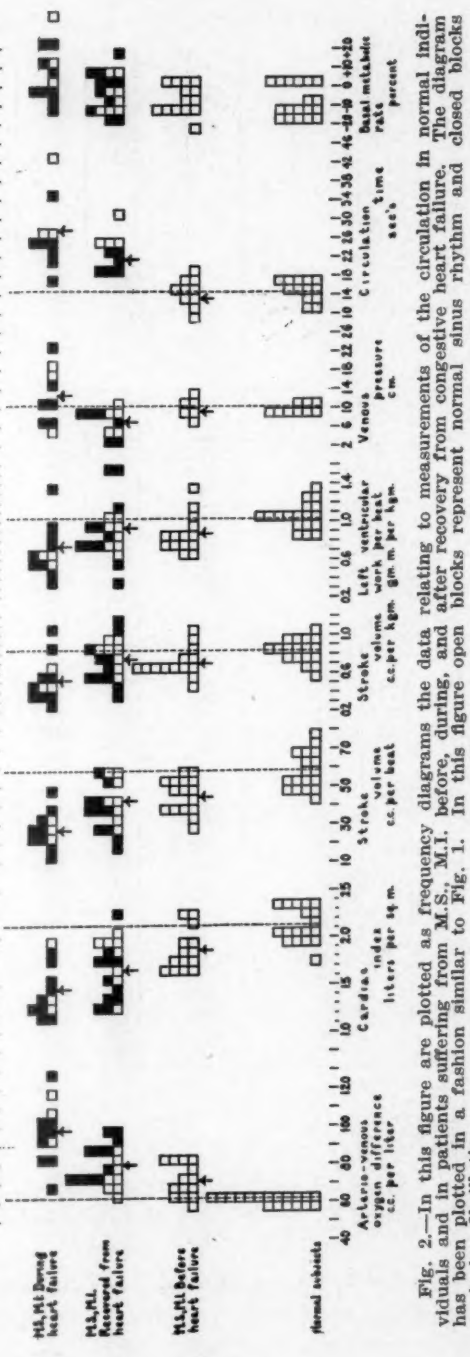

Fig. 2.—In this figure are plotted as frequency diagrams the data relating to measurements of the circulation in normal individuals and in patients suffering from M.S., M.I. before, during, and after recovery from congestive heart failure. The diagram has been plotted in a fashion similar to Fig. 1. In this figure open blocks represent normal sinus rhythm and closed blocks auricular fibrillation.

TABLE

DATA RELATING TO PATIENTS EXHIBITING MITRAL STENOSIS AND INSUFFI

NAME, HISTORY NUMBER, SEX, AGE	DATE	HEIGHT (CM.)	WEIGHT (KG.)	BODY SURFACE (SQ. M.)	OXYGEN CONSUMPTION (C.C. PER MIN.)	BASAL METABOLIC RATE (%)	ARTERIOVENOUS OXYGEN DIFFERENCE (C.C.)	CARDIAC OUTPUT (LITERS PER MIN.)	CARDIAC OUTPUT (LITERS PER SQ. M. PER MIN.)	HEART RATE (PER MIN.)	STROKE VOLUME (C.C.)	CARDIAC AREA (SQ. CM.)
												DURING CONGESTIVE
												Normal
S. S. No. 123829 ♂ 32 years	2/19/36	166.5	65.0	1.73	245	0	115.3	2.13	1.23	70	30	215.6
W. B. No. 131257 ♀ 32 years	4/28/36	151.0	53.8	1.48	207	+14	106.3	1.95	1.32	80	24	194.2
W. H. No. 64020 ♂ 24 years	9/23/35	166.0	44.9	1.47	263	+41	92.8	2.83	1.93	110	28	220.5
												Auricular
J. G. No. 16102 ♂ 28 years	3/13/35	163.0	54.3	1.57	272	+24	95.2	2.86	1.82	102	28	164.3
L. C. No. 36623 ♀ 30 years	3/ 4/35	165.2	60.3	1.66	222	+ 8	96.8	2.29	1.38	100	23	202.9
M. C. No. 119155 ♀ 53 years	1/28/36	147.7	67.3	1.61	207	+14	102.9	2.01	1.25	100	20	194.2
G. MacF. No. 124205 ♂ 57 years	2/25/36	163.0	49.9	1.52	189	- 2	84.2	2.24	1.47	64	27	158.6
J. M. No. 38715 ♂ 37 years	2/27/34	170.3	60.6	1.72	203	-14	100.5	2.02	1.17	76	27	218.0
M. P. No. 62815 ♀ 41 years	4/30/34	161.2	53.1	1.54	180	- 6	90.6	1.98	1.28	124	16	173.2
B. D. No. 59522‡ ♀ 49 years	3/12/36	160.0	46.7	1.46	180	- 2	82.0	2.20	1.51	46	48	148.6
S. C. No. 69400‡ ♀ 39 years	3/23/36	150.1	47.1	1.41	166	- 4	67.8	2.45	1.74	64	38	153.6
C. C. No. 75892 ♀ 44 years	10/25/34	162.5	46.8	1.47	218	+22	125.6	1.74	1.18	142	12	136.1

*In this column the following abbreviations are used:
Rh. = rheumatic fever
hypt. = hypertension
M.S. = mitral stenosis
M.I. = mitral insufficiency
E.H. = enlargement of heart
sl. = slight

†0, +, ±, ↓, ↑, = absent, present, doubtful, decreased, increased, respectively.

‡This patient, unlike the others in this group, was under the influence of digitalis when the special studies of the circulation were made.

order was the same. It is apparent that the presence of these valve lesions usually decreases the capacity of the heart to pump blood, but A. I. seems to have a beneficial influence on M. S., M. I. and results in less impairment of the circulation than is found in M. S., M. I. alone. There is a notion that the development of hypertension in a

IV

CIENCY AFTER RECOVERY FROM AND DURING CONGESTIVE HEART FAILURE

CARDIAC VOLUME (C.C.)	ARTERIAL PRESSURE (MM. HG)	CIRCULATION TIME (SEC.)	VENOUS PRESSURE (CM.)	VITAL CAPACITY (C.C.)	LEFT VENTRICULAR WORK (GM.M. PER BEAT)	STROKE VOLUME PER KG. (C.C.)	LEFT VENTRICULAR WORK PER BEAT PER KG. (GM. M.)	DIAGNOSIS*	EVIDENCE OF CONGESTIVE HEART FAILURE†				
									DYSPNEA	CYANOSIS	RALES	LIVER	EDEMA
HEART FAILURE													
Rhythm													
1528	114/76	42.6	18.1	3200	39.0	0.46	0.60	Rh., M.S. & M.I., E.H.	0	+	+	+	0
1317	110/80	27.0	17.0	1500	31.0	0.45	0.58	Rh., M.S. & M.I., E.H.	+	+	+	+	+
1579	94/70	26.6	5.8	2700	29.0	0.63	0.65	Rh., M.S. & M.I., E.H.	+	+	0	+	0
Fibrillation													
1019	150-130/90	25.4	23.2	2600	43.8	0.52	0.81	Unk., M.S. & M.I., E.H.	+	+	+	+	+
1399	120-110/74	22.5	6.6	2750	31.3	0.38	0.52	Rh., M.S. & M.I., E.H.	+	+	0	+	+
1320	170/100	34.0	11.4	1550	36.7	0.30	0.55	Rh., M.S. & M.I., hypt., E.H.	0	+	+	+	+
965	110/90	24.0	7.9	1750	36.7	0.54	0.74	Rh., M.S. & M.I., E.H.	0	+	+	+	0
1554	140/80	-	-	2700	40.4	0.45	0.67	Rh., M.S. & M.I., E.H.	+	+	+	+	0
1102	112/70	-	-	2080	19.8	0.30	0.37	Rh., M.S. & M.I., E.H.	0	0	+	+	0
876	122/70	25.4	10.3	2000	62.6	1.03	1.34	Unk., M.S. & M.I., E.H.	+	+	0	+ (ascites)	0
918	108/66	20.2	15.8	2000	45.0	0.81	0.96	Unk., M.S. & M.I., E.H.	+	+	0	+	+
769	150/100	17.0	-	1900	20.7	0.26	0.44	Unk., M.S. & M.I., sl. E.H.	±	+	0	0	0

patient suffering from M. S., M. I. exerts a beneficial effect,[12, 13] probably due to dilatation and enlargement of the left ventricle and stretching of the mitral ring. A. I. may perform a similar function. On the other hand, the combination of A. S. and A. I., or their addition to M. S., M. I., exerts a very unfavorable effect on the circulation. Organic M. I. may result in decrease in functional capacity, but M. S., A. S., and A. I. are not individually incompatible with average circulatory function. In patients with valve lesions who had not had failure, the circulation time did not show any marked change except in the M. S., M. I., A. S., A. I. group.

TABLE

NAME, HISTORY NUMBER, SEX, AGE	DATE	HEIGHT (CM.)	WEIGHT (KG.)	BODY SURFACE (SQ. M.)	OXYGEN CONSUMPTION (C.C. PER MIN.)	BASAL METABOLIC RATE (%)	ARTERIOVENOUS OXYGEN DIFFERENCE (C.C.)	CARDIAC OUTPUT (LITERS PER MIN.)	CARDIAC OUTPUT (LITERS PER SQ. M. PER MIN.)	HEART RATE (PER MIN.)	STROKE VOLUME (C.C.)	CARDIAC AREA (SQ. CM.)
RECOVERED FROM CON												
Normal												
S. S. No. 123829 ♂ 32 years	2/24/36	166.2	60.7	1.68	236	- 1	74.3	3.20	1.90	58	55	172.9
W. B. No.131257 ♀ 32 years	5/ 7/36	151.0	46.0	1.39	166	- 4	82.3	2.02	1.50	48	42	183.4
F. S. No. 89187 ♂ 24 years	4/13/35	172.5	49.4	1.58	195	-11	64.6	3.02	1.91	72	42	162.9
M. R. No. 60143 ♀ 32 years	4/ 4/34	149.0	52.2	1.45	158	-12	89.6	1.76	1.21	60	29	154.4
C. W. No. 88345 ♂ 25 years	12/ 7/35	183.2	71.0	1.92	290	+ 6	73.4	4.00	2.03	70	57	273.6
	12/19/35	184.5	70.8	1.94	286	+ 6	75.8	3.77	1.95	70	54	273.2
L. A. No. 7210 ♀ 24 years	5/11/34	162.5	56.0	1.60	179	-12	68.6	2.61	1.64	70	37	140.6
	5/31/34	163.0	57.9	1.64	194	- 8	65.5	2.95	1.80	93	32	137.7
Auricular												
J. G. No. 16102 ♂ 28 years	3/25/35	162.7	48.0	1.50	247	+18	74.1	3.33	2.22	60	56	146.5
J. G. No. 16102 ♂ 28 years	11/12/35	162.5	52.3	1.55	237	+ 9	86.3	2.75	1.80	66	42	154.3
	11/16/35	153.2	51.5	1.55	226	+ 5	86.2	2.61	1.70	50	52	143.5
L. C. No. 36623 ♀ 30 years	3/ 6/35	165.0	60.3	1.66	207	0	74.4	2.78	1.70	70	40	185.3
M. C. No. 119155 ♀ 53 years	2/14/36	148.0	65.8	1.60	193	+ 6	77.8	2.48	1.55	70	35	168.7
G. MacF. No. 124205 ♂ 57 years	2/29/36	163.3	48.9	1.51	182	- 5	70.8	2.57	1.70	68	38	143.1
M. P. No. 62815 ♀ 41 years	5/10/34	160.5	51.5	1.53	159	-17	74.3	2.14	1.40	59	36	162.0
M. R. No. 19718 ♀ 25 years	4/11/34	164.8	48.9	1.52	191	- 2	96.2	1.98	1.30	86	23	189.8
	4/20/34	165.7	47.7	1.52	180	0	98.2	1.83	1.20	106	17	194.4
J. W. No. 28813 ♂ 28 years	3/ 9/34	163.8	50.2	1.53	188	-10	92.0	2.05	1.34	74	28	-
	3/12/34	163.6	50.3	1.53	171	-18	88.0	1.95	1.27	69	29	-

The patient with coarctation of the aorta* was included because of the functional similarity of this lesion to aortic stenosis. There was also evidence of slight aortic insufficiency. The patient at the time of our examination had no clinical evidence of cardiac insufficiency. Nevertheless, marked changes in the circulation had occurred in the year since Grollman and Ferrigan[14] had studied him. The arteriovenous oxygen difference had increased from 61.0 c.c. to 90.3 c.c., and the cardiac output per minute had decreased from 4.90 liters to

*Earlier observations on this patient were reported by Grollman and Ferrigan[14].

IV—CONT'D

CARDIAC VOLUME (C.C.)	ARTERIAL PRESSURE (MM. HG)	CIRCULATION TIME (SEC.)	VENOUS PRESSURE (CM.)	VITAL CAPACITY (C.C.)	LEFT VENTRICULAR WORK (GM.M. PER BEAT)	STROKE VOLUME PER KG. (C.C.)	LEFT VENTRICULAR WORK PER BEAT PER KG. (GM. M.)	DIAGNOSIS*	EVIDENCE OF CONGESTIVE HEART FAILURE†				
									DYSPNEA	CYANOSIS	RALES	LIVER	EDEMA
GESTIVE HEART FAILURE													
Rhythm													
1097	105/58	31.7	5.8	3450	61.0	0.91	1.00	Rh., M.S. & M.I., E.H.	0	0	0	+	0
1198	98/65	24.0	5.3	2100	47.0	0.91	1.02	Rh., M.S. & M.I., E.H.	0	0	0	0	0
1005	106/66	11.5	9.5	4400	49.0	0.85	0.99	Rh., M.S. & M.I., E.H.	0	0	0	0	0
925	115/55	-	-	2100	34.0	0.56	0.65	Unk., M.S. & M.I., E.H.	±	+	0	0	±
2185	102/54	24.0	10.5	3850	60.5	0.80	0.85	Unk., M.S. & M.I.,	0	0	0	0	0
2185	116/66	25.2	8.8	4100	66.8	0.79	0.94	E.H.	+	0	0	0	0
806	95/60	-	-	2700	39.2	0.66	0.70	Rh., M.S. & M.I.,	±	±	±	±	±
781	120/84	-	-	2610	44.4	0.88	0.77	E.H.	±	±	±	±	±
Fibrillation													
856	134/65	19.5	8.1	3250	76.2	1.17	1.59	Unk., M.S. & M.I., E.H.	0	0	0	0	0
926	135/76	19.9	9.1	2750	60.5	0.80	1.16	Unk., M.S. & M.I.,	0	+	0	+	0
821	152/76	22.1	6.3	2900	80.6	1.01	1.57	E.H.	0	+	0	+	0
1218	118-110/74	20.2	6.4	2700	53.9	0.66	0.90	Rh., M.S. & M.I., E.H.	0	0	0	±	0
1160	150/70	22.2	8.1	2000	52.3	0.53	0.79	Rh., M.S. & M.I., hypt., E.H.	0	0	0	0	0
827	118/62	19.7	2.9	1850	46.5	0.78	0.95	Rh., M.S. & M.I., E.H.	0	0	±	0	0
996	100/70	-	-	2200	41.6	0.70	0.81	Rh., M.S. & M.I., E.H.	0	0	↓	↓	0
1271	98/60	-	-	2100	24.7	0.47	0.51	Rh., M.S. & M.I.,	±	±	±	±	±
1310	98/60	-	-	2310	18.5	0.36	0.39	E.H.	±	±	±	±	±
-	115/80	-	-	2400	37.3	0.56	0.74	Unk., M.S. & M.I.	±	±	±	±	±
-	115/80	-	-	2270	38.7	0.58	0.77		±	±	±	±	±

3.52 liters, the cardiac index from 2.50 liters to 1.93 liters, and the stroke volume from 60 c.c. to 41 c.c. The basal systolic blood pressure had risen, and the diastolic had fallen. The heart was greatly enlarged, and the work per beat was not commensurate with its great size (see p. 498). The patient died of pneumonia one month after our observations were made.

In comparing the observations made on the M. S., M. I. groups before the occurrence of failure, during failure, and after recovery from failure, certain differences were found. There were marked impair-

TABLE V

MEAN VALUES OF MEASUREMENTS OF NORMAL INDIVIDUALS,* AND OF PATIENTS WITH MITRAL STENOSIS AND INSUFFICIENCY, BEFORE FAILURE, RECOVERED FAILURE AND DURING FAILURE (TABLE IV)

	ARTERIO-VENOUS OXYGEN DIFFERENCE (C.C.)	CARDIAC INDEX (LITERS PER MIN.)	STROKE VOLUME (C.C.)	STROKE VOLUME (C.C. PER KG.)	LEFT VENTRICULAR WORK PER BEAT PER KG. (GM. M.)	VENOUS PRESSURE (CM. PHYSIOLOGIC SALINE)	CIRCULATION TIME (SEC.)
Normal Individuals	61.4	2.11	57	0.82	1.04	10.0	14.4
M.S. M.I. Before Failure	71.2	1.86	44	0.70	0.88	9.8	13.2
M.S. M.I. Recovered Failure N.R.	74.0	1.71	51	0.88	0.87	8.0	23.3
M.S. M.I. Recovered Failure A.F.	83.2	1.60	37	0.69	0.93	6.8	20.6
M.S. M.I. Recovered Failure N.R. & A.F.	79.5	1.64	43	0.74	0.90	7.3	21.8
M.S. M.I. During Failure N.R.	105.0	1.49	27	0.51	0.61	13.9	32.1
M.S. M.I. During Failure A.F.	94.0	1.42	27	0.51	0.79	12.5	24.1
M.S. M.I. During Failure N.R. & A.F.	96.6	1.44	27	0.51	0.74	13.0	26.5

*Data from Stewart and Watson.[6]

ment of function when heart failure supervened and improvement with the return of compensation, but the improvement was not sufficient to restore the prefailure level of function; in short, those who recovered from failure took an intermediary position between those who had not experienced failure and those who were suffering from it. Analysis of the data shows that there is progressive increase in arteriovenous oxygen difference and progressive decrease in cardiac index and stroke volume in going from the "no failure" group, to the "recovered failure" group, to the "during failure" group. The circulation time was prolonged during, and after, recovery from failure, but the venous pressure was elevated during failure only. In other words, the heart which is the seat of mitral stenosis and insufficiency is a less effective pump, and blood moves more slowly, when congestive failure is present than before it occurred. With recovery from failure the functional capacity impoves, but the work of the heart per beat is no longer commensurate with its size. The stroke volume per kilogram and left ventricular work per beat per kilogram were slightly greater after recovery from congestive heart failure than before it occurred. This is probably an effect of digitalis, as will be shown in other reports.[2, 5] Because of alterations in heart size as a consequence of failure, the giving of digitalis, even though it decreases the size of the heart and increases the amount of work it can do, does not restore normal circulatory efficiency. It appears from this analysis as well as from other observations which we have made[1, 2, 5] that heart failure is associated with decrease in cardiac output, slowing of the velocity of blood flow, dilatation of the heart, and decrease in the work of the heart per beat so that it is no longer commensurate with the heart size (see p. 498). In short, in heart failure of the congestive type the heart fails to pump an adequate amount of blood. The figures which we have discussed are averages, and it is seen from the frequency charts that there is overlapping. A certain level of output does not correspond to a given degree of failure in all cases, but this is only to be expected with differing degrees of valvular and myocardial damage and individual variations in the adjustments of the circulatory system and of the whole organism. Although, in general, the conclusions of McGuire, Hauenstein, and Shore[15] are in agreement with ours, theirs do not appear to be based on adequate data. In the first place, they state that "at the time of the determinations of the cardiac output, varying degrees of improvement in symptoms and physical findings had resulted from rest, the administration of digitalis and other therapeutic procedures." Moreover, they include observations relating to pericarditis with effusion. Obstruction to the flow of blood into the heart is quite different from failure of the heart per se.[16] Since their experiments were designed to compare the direct

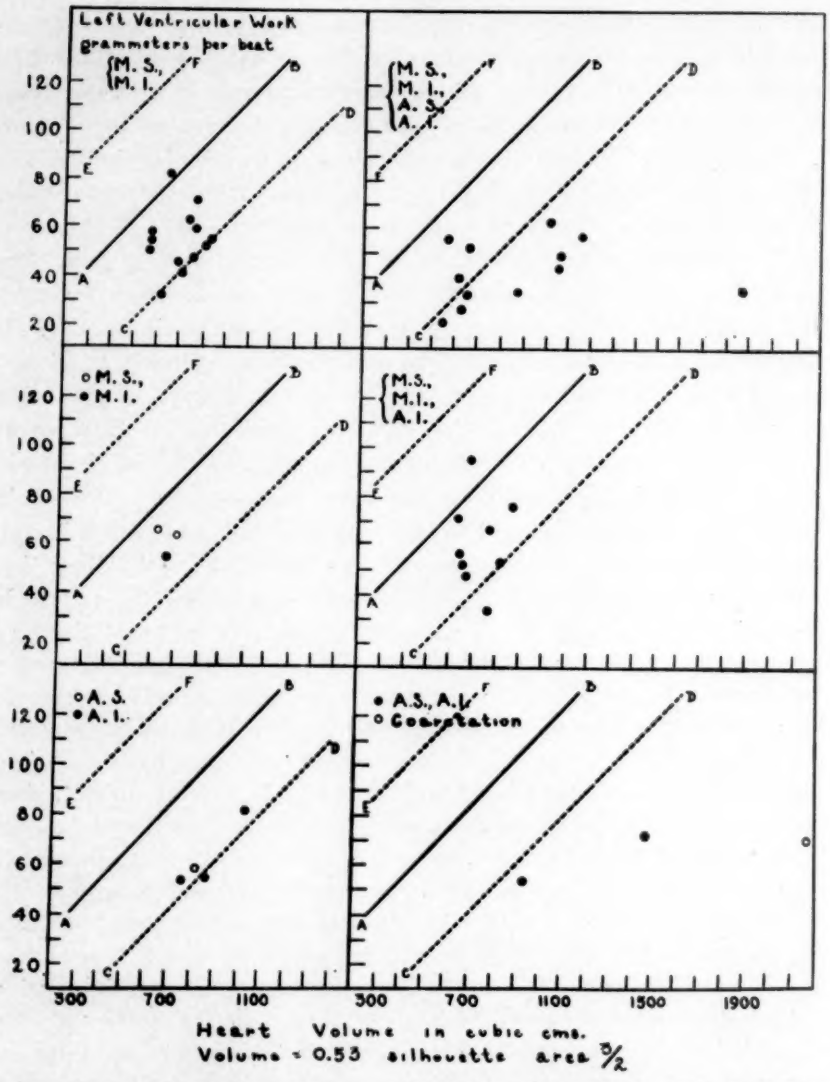

Fig. 3.—Left ventricular work per beat and cardiac volume. The data from Tables II and III relating to work of the left ventricle per beat in the presence of valve lesions are plotted against the corresponding cardiac volumes. Line *AB* represents the best line, the regression of the work on area, defined by Starr, Collins, and Wood on the basis of a statistical treatment of data from a control group of cases. Lines *CD* and *EF* are placed by these authors at a distance of twice the standard deviation from *AB*. It appears from their observations that a patient falling within zone *CD-EF* has a normal circulatory function. That is to say, the work of the heart is commensurate with its size; on the other hand, they found that the values relating to patients who had suffered from cardiac decompensation fell in a zone below *CD*. Each closed or open circle represents a measurement in that valve group as indicated. It is apparent that A.S., A.I., M.S., M.I., and M.S., M.I., lie within the zone *CD-EF* or at the border line *CD*; but A.S., A.I., coarctation, M.S., M.I., A.I., and M.S., A.S., A.I., begin to fall outside the zone of normal circulatory function.

Fick method with the acetylene method, closer correlations would probably have prevailed if comparative measurements had been carried out on the same day, in accordance with the principles established by Baumann and Grollman,[17] rather than at forty-eight-hour intervals.

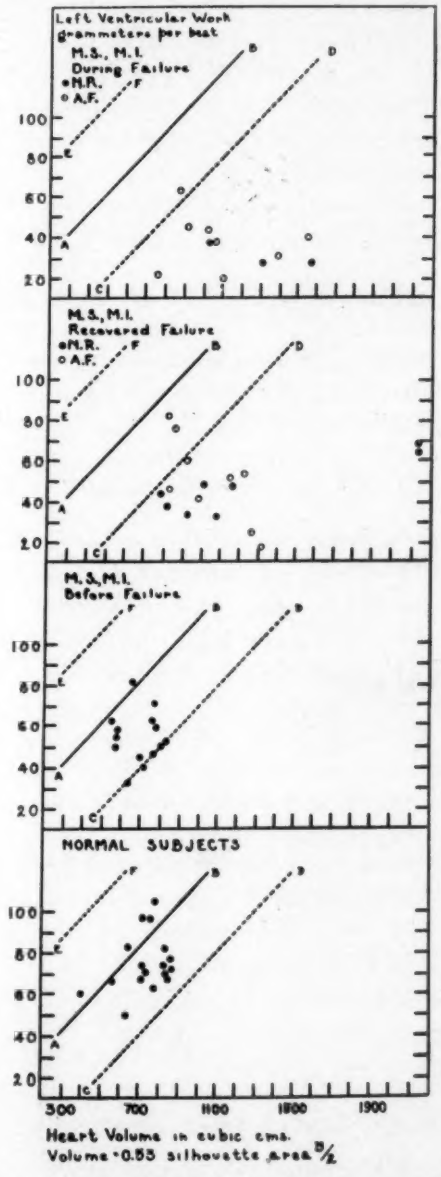

Fig. 4.—Left ventricular work per beat and cardiac volume. The data of Tables IV and V relating to work of the left ventricle per beat are plotted against cardiac volume in normal subjects, in M.S., M.I. before failure, in M.S., M.I. after recovery from failure, and in M.S., M.I. during failure, in a manner similar to Fig. 3. Most of those recovered from failure and during failure fell outside the zone *CD-EF*, but those during failure were for the most part lower and farther away from line *CD*.

The work of the left ventricle per beat has been plotted against the cardiac volume (Fig. 3). Stewart and Cohn[1] suggested that Starling's law[18] of the heart is applicable to human beings with heart failure and that the changes associated with the use of digitalis are subject to interpretation on this basis. Starr and his associates, in a statistical analysis, have shown that in the presence of normal circulatory function the work of the left ventricle per beat is proportional to the size of the heart.[4, 19] From their data they have defined a zone of normal circulatory function. In those falling below their line *CD* the work per beat is not commensurate with the heart size, and patients suffering from congestive heart failure have been found to lie in this area (Starr,[4, 19] Stewart[2, 5]). Work may be calculated by making use of the formula[20] $W = QR + \frac{wV^2}{2g}$, in which W equals work done per beat, Q equals stroke volume, R equals mean arterial blood pressure in mm. Hg $\times$ 13.6, V equals velocity of blood at aorta, w equals weight of blood, and g equals acceleration due to gravity. The last part of the formula $\frac{(wV^2)}{(2g)}$ has been omitted.

With single lesions (aortic stenosis, or aortic insufficiency, or mitral stenosis, or mitral insufficiency) the work done may be commensurate with the size of the heart. Also, in the cases of mitral stenosis and insufficiency all observations lie inside or just at the border of the normal zone *CD-EF* (Fig. 3). In the cases of mitral stenosis and insufficiency and aortic insufficiency, only one observation lies outside the normal zone below line *CD*. In aortic stenosis and aortic insufficiency and coarctation the observations lie in the heart failure zone below *CD*, and in mitral stenosis and insufficiency and aortic stenosis and insufficiency only 3 observations lie in the normal zone *CD-EF*, showing that in 10 instances the work done by the left ventricle was not commensurate with the cardiac size. It appears that aortic stenosis when combined with other lesions results in enlargement out of proportion to the work which the heart can do.

The work relationships for the mitral stenosis and insufficiency groups have been studied. As has already been shown, before the occurrence of failure the left ventricular work per beat may be commensurate with the size of the heart, which places the cases in the normal zone *(CD-EF)*, but for the most part below the best line *AB* (Fig. 4). Of the observations made on these patients after they had recovered from congestive heart failure, two lie in the normal zone and all the others in the heart failure zone. Only one observation made during failure was in the normal zone; all the others lay outside below line *CD*, and farther away from it than those on the patients

who had recovered from failure. In short, the disproportion between work per beat and the size of the heart becomes greater during failure and is not entirely abolished after recovery from failure.

It is likely that the deficiencies which have been demonstrated with the patients in the basal state attain greater proportions when they are active.

CONCLUSION

Valve lesions result in certain defects in the functional capacity of the heart which can be detected before failure is apparent clinically. Single lesions are not incompatible with a fairly normal circulation at rest, but in all instances in which there is more than one lesion functional alterations appear. Aortic stenosis in combination with other lesions results in marked decrease in function. The order of magnitude of the functional defect increased progressively in going from the M. S., M. I., A. I. group to the M. S., M. I. group, to the M. S., M. I., A. S., A. I. group. It is inferred that aortic insufficiency is of functional benefit when superimposed upon M. S., M. I. Patients suffering from M. S., M. I., A. S., A. I. exhibit the most marked alterations. Aortic stenosis results in so much enlargement of the heart that when combined with other lesions the work per beat no longer is commensurate with the size of the organ.

When patients with mitral stenosis and insufficiency suffer failure, all of the measurements of the circulation which we made become subnormal, i.e., the arteriovenous oxygen difference increases, the cardiac index decreases, the circulation time increases, the venous pressure rises, and the work is no longer commensurate with the size of the heart. When they recover from heart failure the functional capacity increases, but usually does not regain its prefailure level.

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Department of Clinical Reports

RIGHT-SIDED PLEURAL EFFUSION IN HEART FAILURE*

Report of an Unusual Case

Curtis F. Garvin, M.D.
Cleveland, Ohio

THE following case is reported in order to attract attention to the fact that right-sided pleural effusion may dominate the picture of heart failure to such a degree that there is difficulty in making a correct etiological diagnosis. The number of aspirations and the total amount of fluid removed are also unique and noteworthy. Furthermore, the pathologic findings are interesting, clear-cut, and definitely indicate that the peculiar clinical manifestations were due to heart failure.

Case Report.—F. Z., a 53-year-old white American male, entered the Cleveland City Hospital, June 24, 1936. His chief complaint was breathlessness. The patient said that he had been in excellent health until Feb. 3, 1936 (about four and one-half months before admission), when he suddenly developed rather marked shortness of breath and slight pain in the precordial region. The pain disappeared shortly, but thereafter the patient had had progressively severe shortness of breath on exertion, weakness, a twenty-pound weight loss, lassitude, and cough productive of mucus.

Examination showed the patient to be normally developed, chronically ill, and moderately dyspneic. The fundi showed slight arteriolar sclerosis. The anteroposterior diameter of the chest was greater than normal. There were signs of fluid in the right pleural space. The heart was enlarged and showed tachycardia and an apical systolic murmur. The blood pressure was 114/80. It was specifically noted that the patient did not have distention of the jugular veins, an enlarged tender liver, or edema of the legs or sacral region.

Urinalysis on numerous occasions gave negative results. The value for the hemoglobin was 70 per cent; the erythrocytes numbered 3,150,000 and the leucocytes 8,400 per cubic millimeter. The value for blood urea nitrogen was 16.8 mg. per 100 c.c. The value for the total serum nitrogen was found to be 815 mg. per 100 c.c., for total serum protein 4.9 gm. per 100 c.c., for serum albumin 2.5 gm. per 100 c.c., and for serum globulin 2.4 gm. per 100 c.c. The albumin-globulin ratio was 1.05. The Kline test for syphilis was negative.

Fluoroscopic and roentgenographic studies of the chest showed the heart to be enlarged in its transverse diameter. The cardiac configuration was of the aortic type. There was cloudiness at the right base due to the presence of fluid. The lung markings were increased in density throughout both lung fields.

The electrocardiogram in the conventional leads showed splintered Q-waves in Leads II and III. The T-wave in Lead III was negative (inverted). Lead IV (made with one electrode at the apex and the indifferent one on the left leg) revealed a positive (upright) T-wave.

*From the Department of Medicine of Cleveland City Hospital and Western Reserve University School of Medicine.
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Bronchoscopy, which was done on June 27, 1936, revealed no abnormalities.

On admission, thoracentesis on the right yielded 1,000 c.c. of serosanguineous fluid. This fluid in the right pleural space reaccumulated almost immediately. Between June 24, 1936, and Sept. 21, 1936, a period of ninety days, thoracentesis was performed forty-five times. The total amount obtained was 47,925 c.c., or an average of 1,065 c.c. on each occasion. For one period of nineteen consecutive days thoracentesis was performed once each day, with removal of 1,000 c.c. every time. The fluid varied in character, being serous or serosanguineous. The specific gravity originally was 1.020, but gradually dropped to between 1.012 and 1.015. Cultures of the fluid for pyogenic organisms showed no growth. The fluid was injected into a guinea pig, which failed to develop tuberculosis. Tumor cells were not present in the aspirated material.

During this period the patient's general condition remained much the same. He showed a constant tachycardia and more or less breathlessness. Fluid never accumulated in the left pleural space to any degree nor did the patient show any more than very slight edema of the feet or sacral region. After Sept. 21, 1936, the patient improved very slowly and thoracentesis was no longer necessary, although the patient did require salyrgan periodically. Recovery, however, was very slow and it was not until June 15, 1937 (almost one year after admission) that the patient was discharged.

The patient got along fairly well thereafter until October, 1937, when he developed severe dyspnea and weakness. At this time his private physician aspirated the right pleural space and obtained 300 c.c. of serous fluid. The patient became steadily worse, however, and was readmitted to the hospital Nov. 2, 1937. The findings were much as before except that the cardiac mechanism was now auricular fibrillation. The right pleural space again contained fluid. There was no edema of the legs. Thoracentesis was performed twice with removal of 500 c.c. of serosanguineous fluid on each occasion. The specific gravity was 1.012. The patient's condition became progressively worse despite all treatment, and he died Nov. 11, 1937.

The diagnoses were: (1) Coronary artery disease; (2) coronary thrombosis, remote, with old myocardial infarct; (3) cardiac hypertrophy and dilatation; (4) myocardial insufficiency; (5) auricular fibrillation; (6) right-sided pleural effusion.

Autopsy.—The autopsy was performed two hours after death. External examination was negative. On section there was no ascites or peritonitis, and no hydrothorax on the left. The right lung showed marked atelectasis. The right pleural space was completely obliterated by fibrous adhesions except for a space above the diaphragm 8 cm. in length. This cavity did not contain fluid. The wall was formed by fibrous tissue covered by a small amount of dried blood. There was no evidence of tuberculosis or tumor.

The heart was markedly enlarged in all directions and weighed 725 gm. The perietal pericardium was adherent to the epicardium over the dorsal aspect of the left ventricle and in the region of the pulmonary conus. The right atrium was dilated and the right auricular appendage contained several large pre-mortem thrombi which were adherent to the wall. The tricuspid valve was normal. The right ventricle showed hypertrophy and dilatation. The pulmonic valve was normal.

The left atrium was dilated; the mitral valve showed slight thickening. One chorda tendinea of the posterior papillary muscle was ruptured. The left ventricle showed hypertrophy and dilatation, with moderate fibrosis of the myocardium. The posterior wall of the left ventricle was the seat of a saccular aneurysm, the opening of which measured 4.5 by 3.5 cm. and the depth of which was 2.5 cm. The wall of this aneurysm bulged posteriorly and consisted of glistening, white fibrous tissue partially covered by friable pinkish-gray thrombi.

The right coronary artery showed moderate to severe arteriosclerosis with reduction of its lumen in some places to one-third of the normal size, but it was not actually occluded. The left coronary artery showed a similar degree of sclerosis, and, in addition, the left circumflex branch was completely occluded save in its proximal 2 cm. This occluded artery led directly into the area occupied by the cardiac aneurysm.

The left lung showed passive hyperemia and emphysema. The right lung was markedly reduced in size, atelectatic, and moderately emphysematous; the pleura was uniformly thickened; and almost the entire moddle lobe was the seat of infarction. The lower lobe contained an infarct which measured 3 by 1 cm. in its greatest diameter. The main branch of the right pulmonary artery was practically occluded by a laminated pre-mortem thrombus which extended into the tributaries leading to the right middle and right lower lobes. The pulmonary arteries showed moderately severe arteriosclerosis.

The kidneys showed slight arterial and arteriolar nephrosclerosis. The remaining organs showed passive hyperemia but no other important abnormality to macroscopic examination.

Microscopic examination of the aneurysm of the left ventricle showed that the wall was made up largely of dense fibrous connective tissue with only an occasional hypertrophied muscle cell. The parietal pericardium was adherent to the epicardium and contained numerous large blood vessels. The aneurysm was lined by a partially organized mural thrombus.

A section of the left circumflex coronary artery showed severe arteriosclerosis and complete fibrous occlusion of the lumen with one to three small channels of canalization.

Microscopically, the middle and lower lobes of the right lung showed atelectasis, emphysema, and infarction. The arteries showed moderate arteriosclerosis. The pleura on the right was thickened and fibrous, with no evidence of acute or chronic inflammation, tuberculosis or tumor. The remaining viscera showed passive hyperemia.

The autopsy diagnoses were: (1) Coronary arteriosclerosis, severe, with occlusion (old) of left circumflex coronary artery; (2) saccular aneurysm of posterior wall of left ventricle near base; (3) cardiac hypertrophy and dilatation (725 gm.); (4) embolism and infarction of right lung; (5) mural thrombosis of right auricular appendage and left ventricle; (6) rupture of chorda tendinea to posterior papillary muscle; (7) chronic adhesive pericarditis, localized; (8) induration collapse of right lung; (9) fibrosis of myocardium of left ventricle; (10) chronic passive hyperemia of liver, spleen, and kidneys; (11) pulmonary emphysema; (12) pulmonary arteriosclerosis, moderately severe; (13) arterial and arteriolar nephrosclerosis, slight.

COMMENT

It has been known for a long time that heart failure causes effusions in the right pleural space more often than in the left.* This case is remarkable, however, in the degree to which right pleural effusion dominated the picture of heart failure. Forty-five aspirations of the right pleural space were done in ninety days, and an average of 1,065 c.c. was removed on each occasion. It is to be noted that for all practical purposes the fluid accumulated in the right pleural space only. Even

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terminally the patient had very slight edema and he never had ascites or a left-sided hydrothorax of any consequence. In fact, the only other significant signs of myocardial insufficiency were breathlessness and tachycardia, and these originally were ascribed to the pleural effusion.

This case accordingly illustrates that at least in its earlier stages heart failure may be evidenced by pleural effusion alone. Fluid in the pleural space under such circumstances raises a problem in differential diagnosis. In this instance the rapid accumulation of this fluid led to an original diagnosis of carcinoma metastatic to the pleura, and the heart disease was considered to be incidental. This possibility was rendered more likely by the fact that the pleural fluid was serosanguineous and had a specific gravity of 1.020. It was this incorrect diagnosis of carcinoma that led to the performance of bronchoscopy. In fact, only upon continued observation and investigation was carcinoma ruled out as a cause of the accumulation of the fluid. It has been noted that the specific gravity gradually decreased until it was that of a transudate (1.012 to 1.015). Whether these changes in specific gravity were significant is not known.

At autopsy, the pleural space on the right was small, and aspiration shortly before death yielded only 500 c.c. of fluid. This was in marked contrast to the amounts obtained only a little over one year prior to death (on one occasion 1,850 c.c. were removed). Evidently the long-standing pleural effusion had led to a sterile pleuritis with the development of pleural adhesions.

The heart was greatly hypertrophied (725 gm.). There are no data as to whether the patient had hypertension at an earlier date, but during the last one and one-half years of his life the blood pressure was always normal. Anatomically, definite evidence of chronic hypertension was lacking. The patient did have severe coronary artery disease with occlusion of the left circumflex branch which resulted in a myocardial infarct at the base of the left ventricle. There was electrocardiographic evidence of this infarct one and one-half years prior to death, and the history indicates that it occurred almost two years before death. A focal pericarditis and a saccular aneurysm had developed at the site of infarction.

These cardiac findings and the presence of severe chronic passive hyperemia of the parenchymatous organs constitute anatomic evidence that the heart had been inadequate for a long time. The sequence of clinical events resulting in death can unquestionably be regarded as being due to cardiac failure.

CHOLESTEROL PERICARDITIS*

Report of a Case

Arthur J. Merrill, M.D.
Atlanta, Ga.

CHOLESTEROL pericarditis is an entity unknown in the English literature. Only a few cases have been reported, all of which appeared in the German literature. The following case is reported because of the rarity of this condition, the remarkable response to treatment, and the possibility that it represents an unusual myxedematous syndrome with an elevated basal metabolic rate.

CASE REPORT

Mrs. L. M., a 54-year-old white woman, was admitted to the Vanderbilt University Hospital Feb. 17, 1937, and discharged March 3, 1937. Her general health had always been good, and her past history was not remarkable save for one miscarriage (of a four-month-old fetus) after bearing four normal children at term. She denied having venereal disease, and there was no family history of tuberculosis. Her mother, who is living, has diabetes mellitus.

A relative stated that the patient had had dyspnea on exertion and dependent edema for eight years, but the patient herself first became aware of her dyspnea in the spring of 1934. This progressed gradually until September, 1936, when she began to have mild attacks of vertigo, slight dyspnea at rest, dependent edema, and swelling of her abdomen. She consulted her family physician who told her that she had hypertensive heart disease and sent her to a hospital where she improved moderately on a regime of digitalis, diuretics, and bed rest. She continued to have dyspnea after discharge and for eleven weeks spent most of her time in bed, during which period her edema and ascites progressed, and she became markedly orthopneic. In October, 1936, she was again treated in the hospital, with little relief. Within the next four months all signs of decompensation became more marked, and early in February, 1937, an abdominal paracentesis was performed. Five liters of clear, straw-colored fluid were removed, after which her dyspnea was relieved for three days, but it returned so rapidly that she was sent to the Vanderbilt Hospital for study and treatment under the direction of Dr. Tinsley Harrison. She had never been unduly susceptible to cold weather, but had noticed that her voice had become more masculine and her skin somewhat drier during the three years previous to admission.

On physical examination her skin was dry, rough, and pale, her voice was low pitched and husky, her features were coarse, and the lateral portion of her eyebrows very thin. Her hair, however, was of normal luxuriance. The heart sounds were distant, but there was no murmur, friction rub, or gallop. The pulse rate was 72 per minute, and the cardiac rhythm was normal. A moderate number of congestive râles were present at both lung bases, more in the left. Very little sclerosis of the peripheral vessels was noted. The arterial pressure, which was 240/140, varied

*From the Department of Medicine, Vanderbilt University.
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TABLE I

	DATE														
	2/17	2/18*	2/19*†	2/20	2/21	2/22	2/23	2/24	2/25	2/26	2/27	2/28	3/1	3/2	3/3
Weight (lb.)	185	186½	177½	172½	170	158	153½	155	153½	151	151	148½	147½	146	146
Blood pressure		210/120	160/90	135/60			140/80	160/80	160/80	140/80					
Basal metabolic rate						Plus 19	Plus 20							Plus 39	
Medicinal preparations		Theocin Salyrgan	Thyroid started		Salyrgan	Theocin									

*Karell diet.
†Pericardial tap.

between inspiration and expiration through a range of 15 mm., but the pulse was not definitely paradoxical. The ascites prevented satisfactory palpation of the liver. Fluoroscopic examination revealed a high diaphragm and a cardiac shadow which was tremendously enlarged to the right and left. There was some increase in the width of the shadow at the base when the patient assumed the recumbent posture.

The following day, 500 c.c. of satiny, opalescent fluid were removed from the pericardial sac by the right thoracic approach. The specific gravity was 1.020 (corrected for temperature), and there were 75 cells per cubic millimeter, chiefly lymphocytes and erythrocytes. Culture showed a gram-positive bacillus after nineteen days which could not be identified (contaminant?), and the guinea pig which was inoculated did not develop tuberculosis after three months. Microscopically, the fluid was found to contain innumerable cholesterin crystals. A quantitative determination of the cholesterol content was not done, but similar fluids described in the literature on cholesterol pleural effusions were found to contain approximately 2000 mg. per cent. The blood cholesterol was 278 mg. per cent. The patient was greatly improved symptomatically after the tap; her arterial pressure fell from 250/115 to 130/65, but she was still in a critical condition. Because of her myxedematous appearance, low-pitched voice, and high blood cholesterol, it was decided to give her ½ grain of desiccated thyroid gland twice a day in spite of the basal metabolic rate of plus 19 (corrected for edema). Table I summarizes the patient's subsequent course; she improved rapidly during the remainder of her stay.

Repeated examinations of the urine showed nothing remarkable save for a small amount of albumin. The erythrocyte and total and differential leucocyte counts were within normal limits, and the hemoglobin was 11.5 gm. per 100 c.c. of blood. The sugar and nonprotein nitrogen content of the blood were normal. Electrocardiograms showed low voltage in all leads, and one record revealed paroxysmal auricular tachycardia.

The patient was not seen again until July 7, 1937, when she stated that she had discontinued her digitalis and thyroid extract one month previously. She agreed to return for study two weeks later, and was instructed to resume thyroid extract, which she took for only one week because of rapid loss of weight. When she returned she felt less nervous than she had for several years and, although she was somewhat weak, she had experienced no dyspnea, edema, or precordial pain. Her weight was only 115 pounds. The lungs presented no evidence of congestion, and there was no enlargement of the liver and no edema. Her voice had a normal pitch. The heart was still markedly enlarged to the left and moderately to the right. Fluoroscopic and physical examinations gave no evidence of pericardial effusion. The arterial pressure was 230/120. The blood cholesterol was 263 mg. per cent, and the basal metabolic rate +6 per cent.

Of the reports of cases of cholesterol pericarditis, only that of Dániel and Puder[1] was accessible. Their patient at autopsy showed lesions in the lung and pericardial sac which were thought to be both syphilitic and tuberculous. The patient's Wassermann was positive, and tubercle bacilli were found in the tissues. However, spirochetes could not be demonstrated and the entire microscopic picture was compatible with that of tuberculosis alone. The authors mentioned one interesting hypothesis in attempting to explain the presence of cholesterol in the pericardial fluid. They thought that originally the patient might have had

hemopericardium, with subsequent hemolysis of the erythrocytes, absorption of part of the fluid, and precipitation of the cholesterol which had been present in the bloody fluid.

We are at a loss to explain the etiology in our case. Even if the patient had definite myxedema, the latter would not account for the cholesterol in the pericardial fluid, for in none of the reported cases of myxedematous pericardial effusion were cholesterol crystals found in the effusion. Although the negative result of the guinea pig inoculation does not rule out tuberculosis, it certainly makes it less likely. Whether or not a bloody effusion might have been present originally cannot be said, but there were very few erythrocytes in the fluid at the time of the tap.

Another feature of this case about which we may speculate is the possibility that the patient had myxedema with an elevated or normal basal metabolic rate. Hurxthal[2] reports several such cases in which there was a favorable response to thyroid extract, and thinks that in the absence of other obvious causes for hypercholesterolemia a patient with this condition should be treated for masked hypothyroidism regardless of the basal metabolic rate. Part of the increase in our patient's oxygen consumption may have been due to her dyspnea, but at the time when the measurements were made none was evident. She was given morphine before the first and second measurements. One should bear in mind that this is merely speculation, as we have no definite evidence that this patient had myxedema.

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Department of Reviews and Abstracts

Selected Abstracts

Scupham, George W., Takats, Geza (de), Van Dellen, Theordore R., and Beck, William C.: Vascular Diseases. A Review of Some of the Recent Literature, With a Critical Review of the Surgical Treatment. Arch. Int. Med. 62: 482, 1938.

In the past year the importance of general involvement in the course of vascular diseases has been emphasized by a number of writers. In the association with this idea, primary vascular hypertension has been occupying a larger place in investigative interest. A brief resume of some of the work on this subject is included in this review.

No attempt has been made to discuss all the papers which have been published, but certain ones have been selected which seem to be contributions to the knowledge of vascular diseases. In some instances material has been included which has been covered, at least in part, in previous reviews. This has been done because the subject seems to bear the emphasis of repetition without becoming commonplace.

This annual review contributes a brief analysis of the literature on this subject. It is an important and useful contribution.

AUTHOR.

Kylin, E., and von Koranyi, A.: Studies on Blood Pressure and Blood Sugar in Rabbits Into Which Pituitary Glands Were Transplanted. Klin. Wchnschr. 17: 668, 1938.

Kylin's theory that a certain number of individuals with hypertension and hyperglycemia are suffering from hyperfunction of the pituitary gland is restated and the antithesis between this group of hypertensive patients and Simmond's disease redrawn. The present study concerns the implantation of the pituitary gland of calves into eight rabbits. Within a few days after transplantation, blood pressure rises (from 75 to 100 mm. Hg) and then falls after one or two months. After six months, however, it rises again and remains elevated at a level of 100 to 115 mm., which is equivalent to a pressure of about 220 mm. Hg in man. Concomitantly, the blood sugar rises from a level of 80 or 100 to 146 mg. per cent. The thesis that hypertensive-diabetic syndrome is due to hyperfunction of the pituitary gland receives additional support.

STEELE.

Gollwitzer-Meier, Kl., HaeuBler, H., and Krüger, E.: Effect of Hydrogen Ion Concentration of Blood on Gaseous Exchange of Heart. Arch. f. d. ges. Physiol. 239: 120, 1937.

An increase in pH following carbon dioxide or lactic acid addition to blood of the heart-lung preparation decreases oxygen consumption. A decrease in pH following sodium carbonate increases oxygen consumption. There is an increase

in mechanical efficiency of the heart when the blood becomes acid and a decrease when it becomes alkaline. The effects of pH are independent of diastolic heart size and constitute a deviation from Starling's law.

KATZ.

Meyer, F., and Eckers, H.: The Action of Tyramin Upon the Circulation, According to Studies in Man. Arch. f. exper. Path. u. Pharmakol. **189**: 200, 1938.

Tyramin acts on intravenous and subcutaneous injection, but not on oral or rectal administration. The quickness with which the rise in arterial pressure passes off, the exceedingly small effect upon diastolic pressure, and the increase in volume output of the heart are all evidence against the notion that tyramin plays a part in maintaining the high arterial tension in essential hypertension.

STEELE.

Rothschuh, K. E.: Diacapillary Hypertension Due to Stasis. Klin. Wchnschr. **17**: 51, 1938.

The author has observed a diastolic arterial hypertension in several patients during periods of congestive heart failure which subsided with recovery. In two examples he shows the regularity with which the level of diastolic arterial pressure follows that of venous pressure. He finds, moreover, that brachial diastolic arterial pressure is on an average 10 mm. Hg higher when the arm is hanging down than when it lies horizontally. This he takes as additional evidence that venous pressure affects diastolic arterial pressure. He concludes, admitting the preliminary nature of this study, that distention of the veins exerts back pressure through capillaries and increases the resistance in the arterioles.

STEELE.

Fasshauer, W., and Oettel, H. J.: A Clinical Contribution on the Variability of Automatic Regulation of the Vasomotor System. Klin. Wchnschr. **17**: 620, 1938.

Arterial pressure was followed in fifty-two normal individuals including some well-trained athletes while the position of the body was changed from 45° with the head up, through the horizontal to 45° with the head down on a tip-table. Arterial pressure was followed by means of Lange's apparatus. Three records are shown. The pressure normally falls when the head is raised and rises when it is lowered. When the arterial pressure is raised either by adrenalin, sympatol, or veritol, the changes in pressure which occur with changes in position are abolished. If the drug does not affect the normal level of pressure, then changes in pressure with change in position are not affected.

STEELE.

Linnell, J. W., Thomson, W. A. R.: Some Cardiological Fallacies. Brit. M. J. **2**: 442, 1938.

The authors apparently have assumed the truth of the old saying, "Not knowing things does not hurt so much, as knowing so many things that are not so." They discuss briefly many unfounded beliefs regarding cardiovascular disease. The viewpoint is interesting and probably helpful in pointing out the frequency with which these beliefs occur.

MCCULLOCH.

Yater, Wallace M.: Pathogenesis of Bundle Branch Block: Review of the Literature; Report of Sixteen Cases with Necropsy and of Six Cases with Detailed Histologic Study of the Conduction System. Arch. Int. Med. 62: 1, 1938.

A review of the essential literature concerning bundle branch block has been made.

Sixteen cases of bundle branch block, with necropsy data, have been reported.

Six cases of bundle branch block studied by means of serial sections through the conduction system have been reported.

Bundle branch block is usually due to disease of the coronary arteries, either rheumatic or degenerative, or to hypertension resulting in strain of the left ventricle and impairment of the nutrition of the endocardium and bundle branch.

Bundle branch block is usually associated with bilateral bundle branch lesions, although one branch is usually more seriously affected than the other and probably usually determines the essential form of the electrocardiographic curve.

The newer, or American, terminology is more nearly correct for bundle branch block in man, although it must be admitted that whether right or left is used to modify the diagnosis of this conduction disturbance, the adjective merely indicates the branch more seriously affected.

The uncommon form of bundle branch block, right bundle branch block, is probably usually due to rheumatic arteritis or rheumatic myocarditis.

The common form of bundle branch block, left bundle branch block, is probably due to degenerative cardiovascular renal disease, meaning coronary arteriosclerosis or arterial hypertension, or both.

A bundle branch need not be entirely destroyed at any level in order to produce electrocardiographic alterations that may be designated as typifying bundle branch block.

An increased amplitude of the ventricular complex is not essential to the electrocardiographic diagnosis of bundle branch block.

Any increase of the QRS interval beyond 0.1 second may indicate lesions of the bundle branches.

Many questions remain unanswered in regard to bundle branch block, and many careful histopathologic studies must be made before most of them can be answered.

AUTHOR.

Ruegsegger, James M.: Pneumococcic Endocarditis. Arch. Int. Med. 62: 388, 1938.

Acute endocarditis occurring in the course of pneumococcemia is probably always caused by the pneumococcus.

Acute endocarditis is a considerably more frequent complication of pneumococcic sepsis than is generally believed.

Pneumococcic endocarditis may be diagnosed ante mortem in about 50 per cent of the cases if certain laboratory facilities are available.

Pneumococcic endocarditis usually occurs as a complication or sequel of pneumococcic pneumonia, runs an acute course, attacks especially the valves of the left side of the heart, is characterized by embolic phenomena, and terminates in the majority of instances in purulent meningitis.

As serum and drug therapy of pneumococcic endocarditis has been almost uniformly unsuccessful, therapeusis should be largely prophylactic, namely, the prevention of bacteremia by means of potent specific serum and the removal or drainage of purulent foci.

AUTHOR.

Stewart, Harold J., Heuer, George J., Deitrick, John E., Crane, Norman F., Watson, Robert F., and Wheeler, Charles H.: Measurements of the Circulation in Constrictive Pericarditis Before and After Operation. J. Clin. Investigation 17: 581, 1938.

In the last two and one-half years the authors have observed nine patients suffering from chronic constrictive pericarditis, and in six of these part of the pericardium has been resected. Studies of the circulation have been made before, as well as after, partial pericardiectomy. This paper records the studies of the circulation together with a statement of their experience with surgical treatment.

Chronic constrictive pericarditis is usually associated with decrease in cardiac output per minute and per beat and decrease in the cardiac index. The venous pressure is elevated and the circulation time prolonged, and there is increase in size and caliber of the peripheral venous channels. Rest in bed and medical therapy may occasion clinical improvement with disappearance of the accumulations of fluid and with changes of the circulation toward normal. After operation in those cured, the measurements assumed normal limits, and in those "improved," the measurements of the circulation approached normal. In this syndrome the symptoms and signs appear to be a consequence of the defects in circulation which the constricting pericardium occasions. These defects appear to be two: (1) obstruction to entrance of blood into the chambers of the heart and (2) interference with contraction and emptying of the heart. These result in (1) decrease in cardiac output per minute and per beat and (2) piling up of blood on the venous side, which accounts for rise in venous pressure and slowing of the velocity of blood flow. Releasing the heart and removing obstruction by resection of part of the pericardium results in return of these functions toward or to normal levels.

AUTHOR.

Krumbhaar, E. B., Ehrich, William E.: Varieties of Single Coronary Artery in Man, Occurring as Isolated Cardiac Anomalies. Am. J. M. Sc. 196: 407, 1938.

Two cases of absence of a coronary artery are reported, both incidental findings at autopsy and apparently causing no damage to the myocardium.

In the first case a large left coronary artery continued around the A-V groove to the anterior surface of the right ventricle, giving off branches that corresponded to those normally given off by both arteries (Hyrtl type of absent coronary).

In the second case, a large right coronary artery supplied most of the heart with conventional branches. Near its origin, however, it gave off one large anomalous branch which passed behind the aorta to supply a good part of the left ventricle, and there was another to the ventricular septum. The possibility must be considered that the former of these represents a true left coronary arising from a misplaced anlage, though the similar cases of Bochkalek and Sanes make this very unlikely.

Other cases of absence of a coronary artery, or possibly misplaced anlage or origin, are tabulated, all but three of which fall into groups corresponding to the types of the two cases reported here.

AUTHOR.

Allan, Warde B., and Baylor, John W.: The Influence of Tonsillectomy Upon the Course of Rheumatic Fever and Rheumatic Heart Disease. A Study of 108 Cases. Bull. Johns Hopkins Hosp. 63: 111, 1938.

One hundred and eight patients subjected to tonsillectomy and adenoidectomy because of rheumatic fever, between 1910 and 1924, were reinvestigated in 1935.

Following operation, recrudescences of acute rheumatic manifestations occurred in 43.5 per cent of the patients.

Recrudescences were common in the first five years after operation. Most of the patients who had repeated recrudescences during this period died of rheumatic heart disease.

Patients living for more than five years after operation had almost as many recrudescences after, as during, the five-year period.

Evidence of continued nasopharyngeal infection was prominent in the patients having recrudescences.

Chorea alone was less often followed by severe rheumatic heart disease than was polyarthritis alone.

Rheumatic heart disease occurred more frequently in those having recrudescences, and deaths from rheumatic heart disease occurred only in this group of patients.

The total incidence of rheumatic heart disease was relatively but perhaps not significantly low.

Since rheumatic heart disease developed in only six of the forty-nine rheumatic patients not having cardiac involvement at the time of operation, it is concluded that tonsillectomy and adenoidectomy are to be recommended in the treatment of rheumatic fever.

AUTHOR.

Stroud, Wm. D., and Shumway, Norman P.: Intermittent Claudication as an Early Symptom of Cardiovascular Disease. Pennsylvania M. J. 41: 894, 1938.

Physicians in various parts of the world are considering the possible relationship of intermittent claudication and cramps in the calves of the legs at night with angina pectoris, coronary occlusion, and essential hypertension.

Studies of patients in the heart clinic of a general hospital and those referred for private cardiovascular consultation during the past ten months suggest: (a) There is some relationship between intermittent claudication and coronary occlusion, since there was an incidence in 57 such cases of 7 (12.2 per cent) with intermittent claudication and only one in a control series of 106 patients without heart disease. (b) There seems to be no definite relationship between hypertension or angina pectoris and patients with intermittent claudication. (c) Patients with angina pectoris or coronary occlusion appear to have no more cramps in the calves of the legs at night or in the muscles of the feet or toes than patients with no heart disease. (d) Patients with hypertension are more likely to have such vascular phenomena in the legs while at rest than are patients with no heart disease.

MONTGOMERY.

Hammer, H. J., and Schulte, T. L.: Changes in Blood Pressure Produced by Prostatic Massage. J. A. M. A. 111: 308, 1938.

Prostatic massage causes a rise in systolic and diastolic pressures in most patients. In about 1 per cent of patients syncope occurs, characterized by vasomotor collapse. This occurred in patients whose average age was 39 years.

The vasomotor response to prostatic massage in the series of 378 patients tested showed no relationship to prostatitis, hypersensitivity to the procedure, hemorrhoids or anal fissures, and various nervous states. The rise in blood pressure following prostatic massage was greatest when the patient was in the lying position. Responses of lesser degree were observed among patients in the bent-over and sitting positions respectively.

A comparison of the response of blood pressure following prostatic massage with the rise from the cold pressor test revealed parallel results among both the patients who had normal blood pressure and those who had essential hypertension.

One should bear in mind the possibility of a considerable rise in blood pressure which occurs with prostatic massage in patients with essential hypertension.

NAIDE.

Fatherree, Thomas J., and Allen, Edgar V.: The Influence of Epinephrine on the Digital Arterioles of Man: A Study of the Vasoconstrictor Effects. J. Clin. Investigation **17**: 109, 1938.

The demonstration of the vasoconstrictor effect of epinephrine on the digital arterioles requires sufficient vasodilatation which may be obtained satisfactorily in the upper extremities (and at times in the lower extremities also) by placing a heat tent over the trunk. This procedure does not impair the vasoconstrictor effect of epinephrine. However, even under these circumstances the injection of epinephrine into the veins of patients with normally innervated extremities may not cause significant vasoconstriction.

For comparative purposes, the temperatures of the skin of the digits of an individual and the temperature of the environmental air must be approximately the same on the different occasions of a study of the effect of injecting epinephrine. Even under these circumstances the results are very variable.

In normally innervated extremities, epinephrine ordinarily produces a slower but more prolonged vasoconstrictor effect in the toes than in the fingers, but there is no constant difference in the magnitude of vasoconstriction, induced by the injection of epinephrine, in the fingers as contrasted with that in the toes.

In subjects with normally innervated extremities, the vasoconstrictor effect of epinephrine on digital arterioles varies widely. Marked variability in the response of the skin temperature of different digits of the same individual and in the response of the skin temperature of the same digit of the same individual on different occasions has been noted. There is some doubt that the response of the temperature of the skin of the digits to the intravenous injection of epinephrine is as reliable a test as has been previously reported.

Patients with vasomotor symptoms suggesting Raynaud's disease do not necessarily possess arterioles which are unduly sensitive to epinephrine. Conversely, great sensitivity of digital arterioles to epinephrine does not necessarily indicate that vasomotor symptoms occur clinically. These observations cast some doubt on the conclusion that the recurrence of vasomotor symptoms after ganglionectomy for Raynaud's disease is due to an increased sensitivity of the arterioles to epinephrine.

AUTHORS.

Davis, Perk Lee: Congenital Absence of the Superficial Volar Arch: An Arteriographic Study. Radiology **31**: 137, 1938.

A case of vasospasm is reported with congenital absence of the superficial volar arch as proved by arteriography.

AUTHOR.

Sandstead, H. R., and Beams, A. J.: Relief of Diabetic Pain of Neurocirculatory Origin by Oral Administration of Sodium Chloride. Arch. Int. Med. **61**: 371, 1938.

Observations were made on thirteen diabetic patients, ten with pain of neuritic origin and three with pain of arteriosclerotic origin. From 11 to 60 gm. of sodium chloride were given for from twenty days to six months. The pain was relieved, and the circulation in most instances was improved.

MONTGOMERY.